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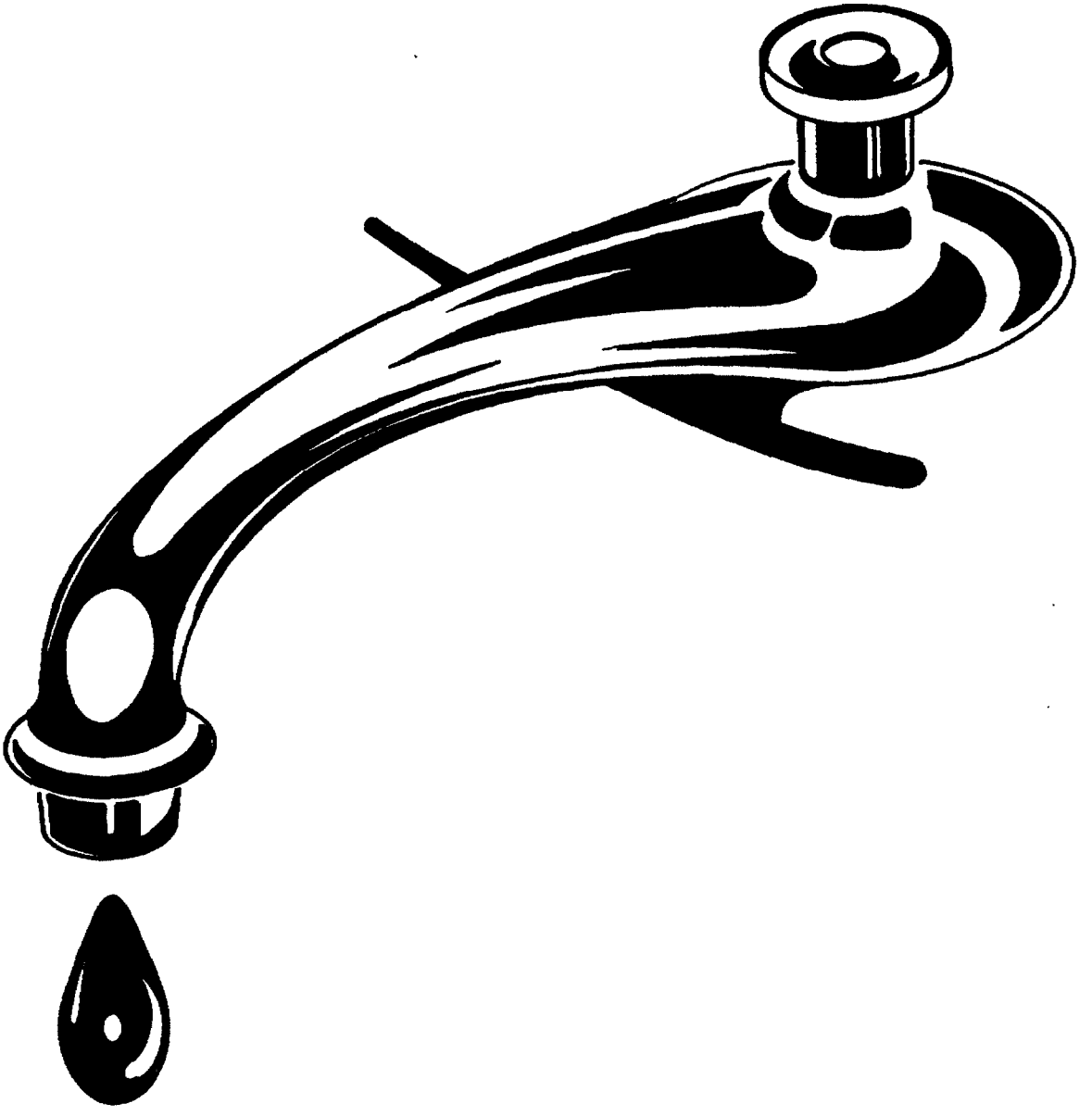
Reducing Lead in Drinking Water:

A Benefit Analysis



REDUCING LEAD IN DRINKING WATER:

A BENEFIT ANALYSIS

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TABLE OF CONTENTS

Table of Contents

Table of Contents

Table of Contents

Table of Contents

LIST OF TABLES

LIST OF TABLES

LIST OF FIGURES

CHAPTER I

INTRODUCTION

This chapter, which provides an overview of the report, consists of three parts: a background of the regulation of lead under the Safe Drinking Water Act, a summary of this analysis of the benefits that could result from a reduction in the amount of lead permitted in U.S. drinking water, and a summary of a case study of the costs and benefits of reducing lead levels in drinking water in the City of Boston.

I.A. Background

The Safe Drinking Water Act (SDWA), passed by the U.S. Congress in 1974, requires the U.S. Environmental Protection Agency (EPA) to protect public health by setting drinking water standards for public water supplies.* Two levels of protection are described in the SDWA. Primary drinking water regulations, applicable only to public water systems, control contamination that may have an adverse effect on human health by setting either a maximum contaminant level (MCL) or a treatment technique requirement. Secondary drinking water standards are non-enforceable recommendations concerning the aesthetic quality of drinking water, e.g., taste or smell.

The National Primary Drinking Water Regulations (NPDWR) were first promulgated at the end of 1975. EPA revises those regulations by setting maximum contaminant level goals (MCLGs)

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* Defined in the Act as water systems serving 25 or more people or having at least 15 service connections.

and related MCLs. MCLGs are non-enforceable health-based goals, intended to protect against known or anticipated adverse health effects, with an adequate margin of safety. MCLS are enforceable limits, to be set as close as feasible to the MCLG; feasibility includes cost and technological constraints. MCLs are proposed at the same time as the MCLGs

On November 13, 1985, EPA proposed National Primary Drinking Water Regulations (NPDWR) to set MCLGs for 28 synthetic organic chemicals, 11 inorganic chemicals, and 4 microbiological parameters in drinking water; these substances are listed in Table I-1. The proposed MCLGs for probable human carcinogens were set at zero, and MCLGS for other substances were based upon chronic toxicity and other data.

Lead is included among the inorganic substances proposed for regulation in the NPDWR. The current MCL for lead is 50 micrograms of lead per liter of drinking water (ug/l);* the proposed MCLG is 20 ug/l.

The 1986 Amendments to the Safe Drinking Water Act contain a provision banning the use of materials containing lead in public water supplies and in residences connected to public water supplies. States have until June 1988 to begin enforcing this ban.

I.B. Summary of Report

This analysis estimates some of the benefits that could result from reducing exposure to lead in community drinking water supplies.

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* This is equivalent to and can be stated alternatively as 0.05 milligrams per liter (mg/l), 0.05 micrograms/gram (ug/g), or 50 parts per billion (ppb).

TABLE I-1. Substances Included in the 1985 Proposed National Primary Drinking Water Regulations (Maximum Contaminant Level Goals)

A. Synthetic Organic Chemicals

1. Acrylamide
2. Alachlor
3. Aldicarb, Aldicarb sulfoxide and Aldicarb sulfone
4. Carbofuran
5. Chlordane
6. Dibromochloropropane
7. o-,m-Dichlorobenzene
8. cis- and trans-1,2 Dichloroethylenes
9. 1,2-Dichloropropane
10. 2,4-D
11. Epichlorohydrin
12. Ethylbenzene
13. Ethylene dibromide
14. Heptachlor and Heptachlor epoxide
15. Lindane
16. Methoxychlor
17. Monochlorobenzene
18. Polychlorinated biphenyls
19. Pentachlorophenol
20. Styrene
21. Toluene
22. Toxaphene
23. 2,4,5-TP
24. Xylene

B. Inorganic Chemicals

1. Arsenic
2. Asbestos
3. Barium
4. Cadmium
5. Chromium
6. Copper
7. Lead
8. Mercury
9. Nitrate and Nitrite
10. Selenium

C. Microbiological Parameters

1. Total Coliform Bacteria
2. Turbidity
3. Giardia
4. Pathogenic Viruses

These benefits are probably much greater than those attributable to just reducing the MCL for lead, but do reflect benefits attainable with reduced exposure to lead through changes in the MCL coupled with changes in EPA's monitoring requirements or other efforts to reduce exposure to lead from drinking water.

There are two primary categories of benefits evaluated in this paper: the public health benefits of reduced lead exposure (Chapters II and IV) and reduced materials damages (Chapter V) relating to the phenomenon of lead's presence in drinking water -- as a corrosion by-product. In addition, because the calculation of health benefits depends on the extent of human exposure, another chapter (Chapter II) presents the available data on the occurrence of lead in public water supplies, and presents estimates of the population exposed to drinking water exceeding the proposed MCLG of 20 ug/l. In assessing the benefits of the proposed reduced lead standard, this analysis assumes that EPA will act to reduce lead levels in tap water, as well as to maintain the current high quality of water leaving the treatment plant. It also relies upon and is sensitive to assumptions about drinking water use and consumption patterns.

This analysis estimates the annual benefits for one sample year, 1988, of lowering the amount of lead permitted from 50 ug/l to 20 ug/l. That one year was chosen because environmental lead levels will have stabilized following EPA's 1984 phasedown of lead in gasoline.*

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* Specifically, this analysis measures effects given the conditions on January 1, 1988, when EPA's proposed ban on leaded gasoline will not yet have taken effect. However, even if EPA promulgates that ban, the estimates in this report will not change significantly.

For comparability, all monetary values are expressed in constant 1985 dollars.* The population baseline is the 219.2 million people served by community water systems.

I.B.1. The Occurrence of Lead in Public Drinking Water

Lead occurs in drinking water primarily as a corrosion by-product; its sources are the materials used in the distribution and residential plumbing systems (of sources as diverse as the US-EPA Air Quality Criteria Document for Lead, 1986; Craun and McCabe, 1975; Kuch and Wagner, 1983; Department of the Environment, 1977; etc). Water leaving the water treatment plant is usually relatively lead-free. However, pipes and solder containing lead are corroded by water, and lead levels at the user tap are often much higher than those found at the treatment plant. While the presence of lead service pipes is relatively restricted geographically in the United States, the use of lead solder (and flux) is ubiquitous. And the combination of copper pipes with solder containing lead found in most residences can result in high lead levels** in first drawn water that has been in contact with the pipe for a period of time -- levels exceeding the current MCL, even with fairly non-corrosive waters (e.g., Nielson, 1976). In particular, newly-installed solder is easily dissolved, and people living in new housing, or in older housing but with new

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* The 1986 Economic Report of the President to Congress (Table B-4).

** This results from galvanic corrosion, which is the corrosion that occurs when 2 metals, with different electro-chemical potential, are in the same environment.

plumbing, are especially at risk of high levels of lead in the drinking water (Sharrett et a1., 1982a; Murrell 1985). Lead concentrations in fully flushed water typical of distribution system water, even under corrosive conditions and with new solder, are generally below 50 ug/l and usually below 20 ug/l.

Because lead occurs generally as a corrosion by-product in U.S. community water supplies, levels in fully-flushed water and in distribution water are typically low. Exposure to lead, however, is from tap water that can contain significant amounts of lead. The estimate of the occurrence of lead in drinking water, therefore, is based upon data collected and analyzed for EPA's Office of Drinking Water in 1979-81. These data portray lead levels partly flushed (30 seconds), kitchen tap samples collected by the Culligan water-softening company.* J. Patterson of the Illinois Institute of Technology analyzed the data. Current evidence indicates that these samples are more representative of consumed water than are the fully-flushed samples taken in compliance with EPA's monitoring regulations. The Culligan data indicate that 16 percent of partly flushed water samples could exceed an MCL of 20 ug/l at the kitchen tap. The findings from this data source are consistent with other analyses of the occurrence of lead in tap water and with studies of lead leaching rates in corrosive and non-corrosive waters.

To this must be added the inhabitants of housing built within the past 24 months and that have plumbing materials containing lead.

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* The use of company names and the presentation of related data does not constitute endorsement of their services.

Many studies have shown that new solder can release significant amounts of lead into water, even exceeding the current MCL of 50 ug/l (e.g., Sharrett et al., 1982a; AWWA-DVGW Cooperative Research Report, 1985). While corrosive waters have the highest lead levels, relatively non-corrosive waters can also leach significant amounts of lead. The highest lead contamination levels occur with the newest solder (i.e., during the first 24 months following installation), but those levels decline and are generally not elevated beyond five years (e.g., Sharrett et al., 1982a; Lassovszky, 1984).

There were 1.7 million new housing starts and permits in the United States during 1983 and 1.8 million in 1984.* Construction data show that housing typically takes six months to a year from permit to potential occupancy, so there are currently about 3.5 million new housing units (i.e., < 24 months). The Statistical Abstract of the United States (1985; Table 58) indicates that the average household contains 2.73 individuals. Multiplied together, a total of 9.6 million people currently live in new housing.

However, not all of these people are served by community water supplies: of the current (1985) U.S. residential population of over 240 million, 219.2 million are served by community water systems and this analysis only addresses that population. In addition, data from the plumbing supply industry show that about

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* Survey of Current Business, U.S. Department of Commerce - Bureau of Economic Analysis, 1985; Table on New Housing Construction.

8 percent* of new plumbing is plastic, so 92 percent of the population has metal pipes. Therefore, the number of people at risk of high lead levels from new solder in new housing is:

$$9.6 \text{ mil} \times \frac{219 \text{ mil}}{240} \times .92 = 8.1 \text{ million.}$$

To calculate the risk to inhabitants of older housing, subtract the number in new housing (8.8 million)** from the total served by community water systems (219.2 million); that indicates that 210.4 million people live in older homes. Based upon the Culligan data, 16 percent of them (33.7 million) are at-risk of high lead levels from partly flushed water at the kitchen tap. Combining the data from Culligan on lead levels in older housing with the new housing exposure estimates indicated that 41.8 million people using public water supplies currently may be exposed to some water that exceeds the proposed MCL of 20 ug/l; we round this to 42 million.

This may be a low estimate

- because it does not include the potential exposure of occupants in housing built within the past 2-5 years (who also probably remain at greater risk of elevated lead levels);***
- because we have not included those who, while living in older housing, have recently had major plumbing repairs and so are also at risk of the potentially high lead levels associated with newly installed solder;

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* This is the average of claims by the Plastic Pipe Institute presented in Mruk (1984) and of the Copper Development Association presented in Anderson (1984).

** Derived: $9.6 \text{ mil} \times \frac{219 \text{ mil}}{240} = 8.8$ million people.

*** Inhabitants of 2-5 year old housing are not included in this analysis because it was not possible to eliminate them from the base and thus avoid double-counting.

- because the Culligan data represent water that is harder than average, whereas high lead levels are often found with soft waters; and

- because the data used are for partially flushed samples, while some people (especially children) may consume water that is closer to first-flush or standing samples (which is more likely to contain higher concentrations of lead).*

In addition, we have not included any data from the estimated 60 million people served wholly or in part by private and non-community water supplies.

There are uncertainties, however, concerning actual patterns of drinking water use and the extent of plastic piping in new construction that could reduce the estimate. Early enforcement of the Safe Drinking Water Act ban on the use of materials containing lead in public water supplies, enforceable in June 1988, could also decrease exposure to lead from drinking water.

The assumptions on the relationship between water lead levels and blood lead levels are taken from the draft (EPA) Water Criteria Document for Lead (1985), which is based upon the recommendations in the Air Quality Criteria Document for Lead (US-EPA, 1986). Those documents assume a linear relationship, at least at the lower blood-lead levels typical of the United States, with different constants for children and adults relating first-flush water lead levels to blood lead concentrations. Those formulae are:

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* Water standing in pipes has a greater opportunity for lead to leach into it and, therefore, is more likely to contain higher lead levels. Many of the factors affecting lead levels in drinking water are discussed in Chapters II and V of this report.

(for children)† PbB* = 0.16** x intake of lead from water

(for adults) PbB* = 0.06** x intake of lead from water.

Alternative assumptions (e.g., those reasonably derived from the results of Richards and Moore, 1982 and 1984) could imply that exposure -- and consequently benefits -- may be underestimated, possibly by several factors.

The estimates of the health benefits associated with this proposed rule rely upon data on the distribution of blood lead levels in children and adults collected as part of the Second (U.S.) National Health and Nutrition Examination Survey (NHANES II), a 10,000 person representative sample of the U.S. non-institutionalized population, aged 6 months to 74 years. That data base is available from the (U.S.) National Center for Health Statistics and analyses of lead-related data from it have been published before (e.g., Annest et al., 1982 and 1983; Mahaffey et al., 1982 ; Pirkle and Annest, 1984).

This analysis uses both point estimates and ranges of blood lead levels associated with specific health outcomes. Other EPA analyses (e.g., US-EPA/ 1986a) use ranges exclusively. Both approaches are supported by the available data.

I.B.2. Benefits of Reducing Children's Exposure to Lead

Elevated blood-lead levels have long been associated with neurotoxicological effects and many other pathological phenomena:

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* PbB = blood lead level

** These constants have a unit of micrograms-of-lead/deciliter-of-blood per microgram-of-lead-in-water/day, or ug/dl per ug/day.

† This formula was derived from Ryu, 1983. An alternative estimate from the data in that paper suggests a coefficient of about 0.4.

an article on lead's neurotoxicity was published as early as 1839, on anemia in the early 1930s, on kidney damage in 1862, and on impaired reproductive function in 1860. As noted in the Air Quality Criteria Document for Lead (US-EPA, 1986), from an historical perspective, lead exposure levels considered acceptable for either occupationally-exposed persons or the general population have been revised downward steadily as more sophisticated bio-medical techniques have shown formerly-unrecognized biological effects, and as concern has increased regarding the medical and social significance of such effects. In the most recent downward revision of maximum safe levels for children, the Centers for Disease Control (CDC) lowered its definition of lead toxicity to 25 micrograms of lead per deciliter of blood (ug/dl, the standard measure of blood lead level) and 35 ug/dl of free erythrocyte protoporphyrin (FEP). As evaluated in the Criteria Document (1986), the present literature shows biological effects as low as 10 ug/dl (for heme biosynthesis) or 15 ug/dl (for certain renal system effects and neurological alterations); indeed, a threshold has not yet been found for some effects (e.g., elevated levels of a potential neurotoxin* or stature effects, Angle et al., 1982; Schwartz et al., 1986).

There is no convincing evidence that lead has any beneficial biological effect in humans (Expert Committee on Trace Metal Essentiality, 1983; and included in the Criteria Document, 1986).

Elevated blood-lead levels have been linked to a wide range of health effects, with particular concern focusing on young

* ALA, or aminolevulinic acid.

children. These effects range from relatively subtle changes in biochemical measurements at 10 ug/dl and below, to severe retardation and even death at very high levels (80-100 ug/dl). Lead can interfere with blood-forming processes, vitamin D metabolism, kidney function, neurological processes and reproductive functions in both males and females. In addition, the negative impact of lead on cognitive performance (as measured by IQ tests, performance in school, and other means) is generally accepted at moderate-to-high blood-lead levels (30 to 40 ug/dl and above), and several studies also provide evidence for possible attentional and IQ deficits, for instance, at levels as low as 10-15 ug/l. Changes in electroencephalogram readings, as another example, have also been observed at these low levels. For many subtle effects, the data may represent the limits of detectability of biochemical or other changes, and not necessarily actual thresholds for effects.

For children's health effects, two categories of benefits were estimated monetarily: 1) the avoidance of costs for medical care for children exceeding the lead toxicity level set by the Centers for Disease Control (i.e., 25 ug/dl, when combined with FEP levels of > 35 ug/dl) and 2) the averting of costs due to lead-induced cognitive effects. Two alternative methods for valuing the potential cognitive damage resulting from exposure to lead were developed. The first of these two alternatives involves assessing the costs of compensatory education to address some of the manifestations of the cognitive damage caused by lead as a

proxy measure for the damage itself. The second relates to one specific indication of that cognitive damage -- potential IQ point loss, and includes a calculation of decreased expected future earnings as a function of IQ point decrement. These estimates neither include many major categories of pathophysiological effects (e.g., renal damage), nor do either the medical costs or the compensatory education costs consider any lasting damage not reversed by medical treatment or compensatory education. These estimates also attribute few benefits to reducing lead levels in children whose blood lead levels would be below 25 ug/dl even in the absence of this proposed rule.

The estimate of reductions in medical care expenses rely upon published recommendations (Piomelli et al., 1984) for follow-up testing and treatment for children with blood lead levels above 25 ug/dl. The costs of such medical services and treatment were estimated at about $950 per child over 25 ug/dl (1985 dollars). This average reflects both lower costs for most of these children and much higher costs for the smaller subset requiring chelation therapy.

The estimates for compensatory education assumed three years of part-time compensatory education (de la Burde and Choate, 1972 and 1975) for 20 percent of the children above 25 ug/dl, averaging about $2,800 (1985 dollars) per child above that blood lead level based upon data from the U.S. Department of Education (Kakalik et al., 1981).

There is extensive literature examining the relationships between IQ, educational levels attained, demographic variables and

earnings (ICF, 1984). The results of that literature were used to estimate the effect of IQ point losses that can occur as a part of the cognitive damage caused by lead exposure upon expected future earnings: one IQ point can directly and indirectly affect earnings by 0.9 percent. The studies of cognitive damage presented in the Air Quality Criteria Document for Lead (U.S. EPA, 1986) show evidence that blood lead levels of 15-30 ug/dl can be associated with IQ losses of 1-2 points, blood lead levels of 30-50 ug/dl can be associated with IQ losses of 4 points, and over 50 ug/dl of blood lead can correlate with losses of 5 points. Data from the Census Bureau on expected future lifetime earnings, deferred for 20 years* at a 5 percent real discount rate and then annualized, yield estimated benefits of avoided damage from reduced exposure to lead. This alternative method for valuing some of lead's cognitive damage indicated that society could save $1,040 per child brought below 15 ug/dl; $2,600 per child brought below 30 ug/dl; and $2,850 per child brought below 50 ug/dl by reducing lead in drinking water (1985 dollars).

In sum, this analysis indicates that the proposed rule could produce benefits of $27.6 million annually in avoided medical expenses; $81.2 million per year in reduced compensatory education costs; and $268.1 million per year in increased lifetime earnings,

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* These costs are deferred because those suffering the effects are children and will not enter the work force for up to 20 years. Obviously, using the largest deferral period (20 years) reduces the value of the benefit and reduces the benefit estimate, whereas 8- or 10-year-old children may begin working within 8 years and so would have a much shorter deferral period. This biases the estimates downward slightly.

based upon sample year 1988; these estimates are in 1985 dollars. Note that compensatory education and affected earnings are alternative methods for valuing aspects of the cognitive damage caused by lead.*

In addition, benefits potentially derived by decreasing the incidence of two other categories of health effects (lead's adverse effect upon children's growth and fetal effects) were not estimated in dollar terms. Assuming that pregnant women are distributed proportionately throughout the country, data from the Census Bureau** on birth rates and demographic distributions indicate that 24 percent of the total population is women of child-bearing age (15-44) and that the birth rate is 67.4 births per 1,000 women aged 15-44. Therefore,

41.8 million x 24% x 67.4 per thousand = 680,000.

It is estimated that this proposed rule could prevent 680,000 fetuses from being exposed to elevated lead levels. The fetal effects are particularly important, because several recent studies have shown that lead exposure within the normal range (6-20 ug/l) can be associated with various negative pregnancy outcomes (such as early membrane rupture and even miscarriages, e.g., Moore 1982; Wibberly et al., 1977), and with low birth weight, inhibited postnatal growth and development (e.g., Bornschein, 1986; Bellinger, 1985 and 1986; Dietrich et al., 1986). In addition, this proposed rule could prevent 82,000 children from risk of growth effects.

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* This also biases the results downward because there is a strong rationale for considering these effects as additive.

** Statistical Abstracts (1986), Tables 27 and 82.

I.B.3. Blood-Pressure-Related Benefits and Other Adult Health Effects

Lead has long been associated with elevated blood pressure, but until recently most of the studies have focused only on hypertension and relatively high lead levels typically found only in those occupationally exposed to lead. Several recent studies, however, (e.g., Pirkle et al., 1985; Harlan et al., 1985; Pocock, 1984 and 1985), have found a continuous relationship between blood lead and blood pressure. These studies provide evidence for a small (compared to other risk factors) but robust relationship after controlling for numerous other factors known to be associated with blood pressure. Experimental animal studies in several species of rats and pigeons also provide evidence of a relationship between moderate blood-lead levels and increases in blood pressure.

To calculate these benefits, logistic regression equations were used to predict how reducing exposure to lead in drinking water would affect the number of hypertensives in the U.S. population. These estimates cover only males aged 40 to 59, because the effect of lead on blood pressure appears to be stronger for men and because the correlation between blood pressure and age is much smaller in this age range, reducing the potential for confounding due to the correlation between blood lead and age. The estimates rely upon 1) site-adjusted coefficients from analyses of the NHANES II data relating blood lead levels to increases in blood pressure* and 2) coefficients relating blood pressure increases to more serious

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* The specific coefficients and the basis for their derivation are described in the Addendum to the Criteria Document, 1986, which is included in volume 1 of that publication.

cardiovascular disease outcomes, based on data from the Framingham Study (McGee et al., 1976) and Pooling Project (1976), as confirmed by Levy et al., 1984.

Levy has demonstrated that the risk coefficients from the Framingham Heart Study, when coupled with the observed reductions in blood pressure, smoking, and cholesterol in the U.S. population during the 1970s, correctly predicts the observed reductions in cardiovascular mortality in the overall population during that decade. The Pooling Project showed that the Framingham coefficients adequately predicted cardiovascular outcomes (such as strokes and heart attacks) in the other five large prospective heart studies performed in the U.S. Therefore, while caution is clearly warranted in view of the limited data on the effect of lowering blood lead levels on blood pressure, use of the regression coefficients from the Framingham Study provide a reasonable basis by which to predict potential changes in cardiovascular outcomes associated with blood pressure changes due to decreased lead exposure.

Based upon this information, reducing exposure to lead from drinking water in 1988 could reduce the number of male hypertensives (aged 40 to 59) by 130,000. Using estimates of the costs of medical care, medication, and lost wages, such a reduction in hypertension incidence would yield a value of $250 per year per case avoided (1985 dollars).

These estimates of how blood pressure reductions would affect the incidence of various cardiovascular diseases were based on projections of changes in blood pressure as a result of the proposed rule and estimates of the relationships between blood pressure and

heart attacks, strokes, and deaths from all causes. As noted earlier, the latter estimates were derived from several large epidemiological studies, primarily the Framingham study. However, because those studies included very few nonwhites, the estimates were further restricted to white males, aged 40 to 59. Thus, the benefits estimates do not include middle-aged, nonwhite males.

The basis of most of the medical costs are the cost-of-illness estimates presented in Hartunian et al., 1981, which were adjusted in three ways to reflect current conditions. First, we inflated them to 1985 dollars using data from the 1986 Economic Report of the President to Congress. Second, we adjusted the costs to reflect changes and improvements in medical treatment, including the tripling in the incidence rate of coronary bypass operations that occurred between 1975 and 1982. Third, Hartunian used a 6 percent real discount rate to present-value future expenditures, while this analysis uses a 10 percent real discount rate.

The value of reductions in heart attacks and strokes was based on the cost of medical care and lost wages for nonfatal cases. Expected fatalities from heart attacks and strokes were included in the estimate of deaths from all causes. That procedure yielded benefits of $65,000 per heart attack avoided and $48,000 per stroke avoided (1985 dollars) for the 240 heart attacks and 80 strokes estimated to likely be avoided in 1988 because of this proposed rule. It is important to note that these estimates do not account for any reductions in the quality of life for the victims of heart attacks and strokes (e.g., the partial paralysis that afflicts many stroke victims).

Valuing reductions in the risk of death is difficult and controversial, with a wide range of estimates in the literature. EPA's policy guidelines (U.S. EPA, 1984c), for example, suggest a range of $400,000 to $7 million per statistical life saved. Using $1 million per case, the benefits of reduced mortality dominate our estimates of total blood-pressure-related benefits; these total $240 million in 1988 for the 240 deaths estimated as likely to be avoided in that year. Altogether, the monetized benefits of reducing adult male exposure to lead in drinking water are estimated to total $291.9 million per year (in 1985 dollars), using 1988 as a sample year.

In addition, because lead crosses the placental barrier and is a fetotoxin, pregnant women exposed to lead are at risk of complications in their pregnancies and damage to the fetus. (Fetal effects are discussed above, under children's health effects.) While we have not monetized any of these reproductive effects, as noted above, 680,000 pregnant women per year probably receive water that exceeds the proposed standard of 20 ug/l, and would benefit from the proposed rule. Lead-induced effects on male reproductive functions have also been discussed in the scientific literature but are not included in this report.

I.B.4. Benefits of Reduced Materials Damage

A third category of monetized benefits relates to the phenomenon of lead's presence in drinking water: it is a product of the corrosive action of water upon the materials of the distribution and residential plumbing. For the most part, therefore, treatment

processes used to reduce high levels of lead in drinking water are the same as treatment processes used to reduce the corrosion potential of the water. Reducing corrosion damage will produce substantial benefits to water utilities, their rate-paying customers, and building owners.

Published estimates of the costs of corrosion damage range from $12 to $46 per person per year (1985 dollars), and are summarized in Table I-2. Estimates of the costs that can be avoided by corrosion control measures range from 20-50 percent of total damage. The point estimate of avoidable corrosion costs (i.e., the benefits of corrosion control) is $8.50 per capita annually (1985 dollars). For comparison, estimates of average corrosion treatment costs range from under $1 per person per year (based upon the experience in 18 cities currently treating their highly corrosive waters) to about $5 per person per year (based upon the highest treatment costs presented in the ODW cost report).* As a point estimate, we assumed per capita annual treatment costs of $3.80 (1985 dollars).

Estimates of the extent of corrosive water also vary. A commonly accepted profile is that developed by the U.S. Geological Survey in the early 1960s, which identified the Northeast, Southeast, and Northwest sections of the country as having the softest and most corrosive waters (Durfor and Becker, 1964a and

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* The range, however, is quite wide and highly sensitive to system size. These represent average costs. In some very small systems (i.e., serving 25-100 people), costs may be many times higher.

TABLE I-2 . Estimates of Annual Per Capita Corrosion Damage (1985 dollars)

Studies	Estimated Annual Corrosion Damage (per capita)			Corrosion Damage Avoidable Through Water Treatment	Annual Per Capita Benefits of Corrosion Control	Assumptions/Notes
	Distribution systems	Residential	Total			
Kennedy Engineers (1973)	$5.57	—	$16.71*	30%*	$5.01*	Assumed 30% potential reduction in corrosion damage and that distribution costs were one-third of total costs.
Hudson & Gilcreas (1976)	$8.68*	—	$26.04*	50%	$13.02*	They did not include increased operating costs. Per capita estimate assumes 200 million people are served by public water systems. Assumed that distribution costs were one-third of total costs.
Kennedy Engineers (1978)	—	$30.87*	$46.30*	20%	$9. 26*	They calculated $6.17 per capita in savings to residence owners. Assumed residential costs were two-thirds of total costs.
Bennett et al. (1979) (cited in Ryder, 1980)	$9.40	—	$28. 20*	20%	$5.64*	Assumed that 200 million people are served by public water systems and that distribution costs were one-third of total costs.
Energy & Environmental Analysis (1979)	$3.98	$7.97	$11.95	38%	$4.54	This is an admitted underestimate: it includes only damage to pipes (not damage to water heaters, increased operating costs, etc.)
Ryder (1980)	$1.17	$22.19	$23.36	25%	$5.84	Ryder ascribed 95% of corrosion damage to private owners.
Kirmeyer & Logsdon (1983)		$23.60*	$35.40*	40%	$14.16*	Assumed residential costs were two-thirds of total damage.
					AVERAGE $8.21 W/OUT EEA $8.82	

* These estimates have been calculated by the authors of this paper. Assumptions are noted above.

1964b). The combined populations of those states are 67.7 million people (1980 census). Assuming that these areas are served proportionately by community water systems,* 61.8 million people would benefit from actions to reduce the corrosivity of their water. That figure, multiplied by $8.50 per person, yields annual benefits from reduced corrosivity of $525.3 million (1985 dollars).

I.B.5. Summary of Annual Benefits of Reduced Lead in Drinking Water

This analysis of the benefits of reducing exposure to lead in drinking water indicates that the monetized annual benefits could range from $926.0 to $1,112.9 million (1985 dollars) for sample year 1988. In addition, there are numerous health benefits of reduced exposure to lead that are not monetized. The annual monetized benefits are summarized in Table I-3, and the non-monetized benefits are presented in Table I-4.

Based upon the latest cost estimates used by the Office of Drinking Water** the projected benefits exceed the costs by about 4:1. Expressed differently, lowering the MCL to 20 ug/l could produce annual net benefits of about $800 million in 1988.

It should be emphasized that considerable uncertainty is associated with these estimated benefits, uncertainties derived both from the current state of knowledge concerning lead health effects and the valuation of avoiding such effects. Other

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* Of the total population of about 240 million, 219.2 million people are served by community water systems.

** These calculations use preliminary EPA Office of Drinking Water cost estimates. Costs and net benefits will be discussed more extensively in other documents associated with this proposed rulemaking.

TABLE I-3. Summary of Estimated Annual Monetized Benefits of Reducing Exposure to Lead from 50 ug/l to 20 ug/l (1985 dollars) for Sample Year 1988

Estimated population exposed to drinking water exceeding preposed MCL	42 million*
Children's health benefits	
-reduced medical costs	$27.6 million
-reduced costs of cognitive damage	
Method 1 - compensatory education	$81.2 million
Method 2 - decreased future earnings	$268.1 million
TOTAL: Method 1	$108.8 million
Method 2	$295.7 million
Adult health benefits (males only)	
-reduced hypertension savings (males, aged 40-59)	$32.5 million
-savings from fewer heart attacks (white males, aged 40-59)	$15.6 million
-savings from fewer strokes (white males, aged 40-59)	$3.8 million
-savings from fewer deaths (white males, aged 40-59)	$240.0 million
TOTAL:	$291.9 million
Materials benefits	
-benefits of reduced corrosion damage	$525.3 million
TOTAL ANNUAL MONETIZED BENEFITS	
-Method 1 - using compensatory education	$926.0 million
-Method 2 - using decreased future earnings	$1,112.9 million
ESTIMATED ANNUAL COSTS	$230.0 million
NET ANNUAL MONETIZED BENEFITS	about $800 million

*Total population served by community water systems: 219 million

TABLE I-4. Summary of Estimated Annual Non-monetized Benefits of Reducing Exposure to Lead from 50 ug/l to 20 ug/l for Sample Year 1988

	Reductions in Numbers of People at Risk
Estimated population exposed to drinking water exceeding proposed MCL	42 million*
Children's health benefits	
-children requiring medical treatment	29,000
-loss of 1-2 IQ points	230,000
4 IQ points	11,000
5 IQ points	100
-children requiring compensatory education	29,000
-children at risk of stature decrement	82,000
-fetuses at risk	680,000
-increased risk of hematological effects	82,400
Adult health benefits	
-cases of hypertension (males, aged 40-59)	130,000
-heart attacks (white males, aged 40-59)	240
-strokes (white males, aged 40-59)	80
-deaths (white males, aged 40-59)	240
-(reduced risk to pregnant women ((women, aged 15-44) (same as fetuses	680,000)))

* Total population served by community water systems: 219 million

analogous efforts to estimate benefits associated with reducing lead in drinking water may be useful in helping to judge how reasonable these present benefit estimates are.

I.C. Boston Case Study

In the spring of 1986, Jonathan Jacobson analyzed the incremental costs and benefits to the City of Boston of reducing the lead MCL from 50 ug/l to 10 ug/l (Jacobson, 1986).* That analysis, carried out as a masters thesis project at Harvard University, focused on Boston as a city with high potential for increased lead exposure via drinking water.

Boston's water is highly corrosive: it is relatively acidic (pH = 6.7) and soft (14 mg/l as $CaCO_3$), and has low alkalinity (Karalekas et al., 1975). Boston also has a large percentage of lead pipes in service. During the 1970s, several studies found high lead levels in Boston's drinking water (e.g., Karalekas et al., 1975; and several internal studies conducted by the Massachusetts Water Resources Authority and the [Boston] Metropolitan District Commission).

To reduce the high lead levels, Boston began corrosion control treatment. Monitoring performed by EPA's Region I from 1975 to 1981 indicated that lead, iron and copper levels dropped significantly (Karalekas et al., 1982). However, while lead concentrations generally decreased to below the current MCL of 50 ug/l, additional treatment will be necessary to comply with a lowered MCL.

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* He assumed a lowered MCL of 10 ug/l because that is the feasibility limit for current treatment and technology.

Jacobson analyzed the incremental annual costs and benefits of additional efforts by Boston to control further the corrosivity of its water, using sample years 1988 and 1992, and estimating all costs in 1985 dollars. His analysis assumed the following:

- compliance will be measured by a standing "grab" sample, that is, a sample taken immediately after turning on the faucet at any random time during the day after an unknown period of standing;
- it will be impossible for every tap to meet the lowered MCL, even using state-of-the-art treatment, and so samples should be averaged; and
- that, while the effectiveness of specific treatment procedures varies in not-yet-well-understood ways when actually used in the field, corrosion control is ultimately feasible with current state-of-the-art methods.

Jacobson, using data from EPA Region I and the Massachusetts Water Resources Authority, calculated the benefits of additional 2-stage treatment for Boston's water: further raising the pH (to reduce the acidity of the water) and installing several pumping stations to maintain a consistent concentration of sodium hydroxide throughout the system.

Jacobson used the same categories of monetized health benefits* as those described in this EPA analysis, except that he did not include the estimates of cognitive damage associated with decreased future earnings. His estimates of materials benefits rely only upon the Kennedy Engineers (1978) study and information in the American Water Works Association Corrosion Manual (AWWA-DVGW, 1985).

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* For blood-pressure-related estimates, however, he used the non-site-adjusted coefficients from the NHANES II contained in Pirkle et al., 1985.

His results, summarized in pages 36-39 of his study, indicate incremental costs of $700,000 per year (using sample year 1988) and incremental annual benefits of $7.9 million (including estimated health benefits of $6.9 million and materials benefits of $950,000; based upon sample year 1988). This yields estimated net annual benefits of $7.2 million and a benefit to cost ratio of 11:1 (compared to the estimate of 4:1 in this analysis).

It is, however, unclear whether or how these results can be extrapolated to other U.S. water systems and cities, and therefore, to this proposed rule.

CHAPTER II

OCCURRENCE OF LEAD IN DRINKING WATER

Lead can contaminate drinking water through several pathways. It can result from naturally present lead in the source water, it can result from contamination of the water supply, or it can result as a by-product of corrosive water.* In the last case, the sources of the lead are the pipes, plumbing fixtures, flux and solder of the distribution system and within private residences or other buildings. Most contamination of drinking water with lead results from the corrosion of materials containing lead.

Section A of this chapter discusses the sources and factors affecting the contamination of drinking water by lead.** Section B of this chapter discusses the available data on the occurrence of lead in community drinking water supplies. Because lead occurs in drinking water primarily as a corrosion by-product, contamination

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* Corrosion is the deterioration of a substance or its properties due to a reaction with its environment. In this paper, the "substance" that deteriorates is the pipe -- whether made of metal, asbestos-cement, cement, or plastic -- and the flux and solder joining the pipes, and the "environment" is water. That is, we are concerned with internal corrosion. (Pipes and other water treatment equipment can also corrode externally.)

** Chapter V also contains a discussion of the relationship between corrosive water and lead in drinking water, but from the perspective of potential corrective action.

levels are higher in tap water in homes* than in the water leaving the water treatment plant or flowing through the water mains. Section C contains EPA's estimates of exposure to lead in the drinking water provided by community water systems in the United States.

The exposure estimate presented in this chapter serves as the basis for calculating the benefits from a potential reduction in exposure due to a lowering of the Maximum Contaminant Level (MCL) for lead from the current 50 micrograms of lead per liter of water (ug/l) to 20 ug/l.**

II.A. Sources of Lead in Drinking Water

The principal source of lead in ambient surface water is anthropogenic lead particulate from the atmosphere, which come mostly from the combustion of leaded gasoline (e.g., Laxen and Harrison, 1977; Trefry et al., 1985) and, to a lesser extent, the smelting of ores and the combustion of fossil fuels. Evidence indicates that much of the lead in surface waters will end up in sedimentary deposits (Laxen and Harrison, 1977), and most of the

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* Lead contamination of drinking water as a result of corrosion also occurs in commercial buildings, schools, etc. There is less information on the factors that determine lead levels in these buildings, however, than on the use patterns and materials in private homes. Therefore, this analysis examines only exposure to lead in residential circumstances. Additional research is needed in other areas of exposure including the work place (factories, office buildings, etc.) and in schools.

** This is equivalent to and can be expressed as 0.020 milligrams per liter (mg/l), 0.020 micrograms per gram (ug/g), or 20 parts per billion (ppb).

lead dissolved in rain will precipitate and be filtered out by the soil. Lead from runoff or fallout will also precipitate and be retained within the soil or sediments.

The source of most lead in ground water is geochemical; that is, the minerals contained in the rocks and soil through which the ground water flows. Concentrations of lead in ground water in the United States are typically under 10 ug/l (Lovering, 1976) and, generally, lead volubility is very low in ground water.

The principal source of lead in drinking water is neither naturally occurring lead in ground water nor anthropogenic lead in surface water, however. It is the materials of the water supply and distribution systems and the plumbing in homes and other buildings. (See sources as diverse as Craun and McCabe, 1975; [U.S.] National Academy of Sciences, 1977; EPA's Lead Technologies and Costs Document, 1984; Kuch and Wagner, 1983; AWWA-DVGW Cooperative Research Report, 1985; EPA's Lead Occurrence Document, 1985; EPA's Air Quality Criteria Document for Lead, 1986; etc.). The highest concentrations of lead are found where pipes or solder containing lead are used in combination with corrosive waters, where water has been sitting for many hours, or where there is newly-installed piping or repaired joints using flux or solder containing lead.

II.A.1. Variables Affecting Lead Levels in Drinking Water

The lead used in service pipes* or as part of lead/tin solder is relatively resistant to corrosion under simple laboratory

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* Service pipes connect the main pipes of the water distribution system to the plumbing contained within the home.

conditions. In addition, it can be protected from corrosion by a thin layer of relatively insoluble film, composed of carbonate compounds of lead or calcium, that forms on the surface of the metal (Patterson and O'Brien, 1979; Schock and Gardels, 1983; Lassovszky, 1984; etc.). Water, however, is a corrosive substance.

In addition, the combination of copper piping with tin/lead solder found in most residences can result in galvanic corrosion,* which can yield lead levels much greater than expected from the simple corrosion of the water alone.

Other conditions typical in private homes exacerbate the results of galvanic corrosion and can contribute to high lead levels in home tap water.** These include the facts that residential plumbing materials are often less corrosion resistant and well-protected than those used in distribution systems (AWWA Committee Report, 1984), that water often remains overnight in household pipes, and that some water used in private homes is heated, greatly increasing its corrosive potential.

II.A.1.a. Key Water Parameters Affecting the Volubility of Lead

All water is corrosive to some degree. However, some qualities of water make it more corrosive for certain materials. The volubility of lead (also called plumbosolvency) is complicated.

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* Galvanic corrosion results when two metals, with different electrochemical potential, are in the same environment.

* The maximum equilibrium level is determined by the specific qualities of the water. Because waters in public drinking systems rarely reach equilibrium levels (for purposes of corrosion), non-equilibrium conditions are assumed in the analysis presented in this document.

Probably the master control variable in the volubility of lead is pH,* although it is closely interrelated with carbonate.** Waters below pH 6.8 are very plumbosolvent as can be waters with pH above 10.2 (e.g., Moore, 1973; Schock, 1980; Sheiham and Jackson, 1981; Murrell, 1985), but pH does not have a strictly linear relationship to lead levels in water (e.g., Patterson and O'Brien, 1979; Pocock, 1980; Schock and Gardels, 1983). In addition, at low or very high carbonate alkalinities,** lead is soluble throughout the pH range of drinking water (e.g., Department of the Environment, 1977; Pocock, 1980; Jackson and Sheiham, 1980; Schock, 1980; Sheiham and Jackson, 1981; Kirmeyer and Logsdon, 1983; Schock and Gardels, 1983; Gregory and Jackson, 1984; AWWA-DVGW Report, 1985). Soft **water†** is usually plumbosolvent, but several studies have shown that very hard water can also be plumbosolvent (e.g., Department of the Environment, 1977;

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* The factors of water that inhibit or enhance corrosion are discussed in Chapter V, including measures of those parameters. In short, pH is a measure of the concentration of hydrogen ions (H^+) in the water, which is important because H^+ is one of the major substances that determines how much metal can be corroded electrochemically.

** The carbonate content (measured indirectly by alkalinity and pH, and usually given in units of equivalent calcium carbonate , $CaCO_3$) relates mostly to the presence of dissolved bicarbonate and carbonate ions in the water and enables the formation of a relatively insoluble protective coating on the inside of the piper forming a barrier between the water and the materials of the plumbing system.

† Water with low levels of calcium and magnesium ions, which can help form a protective coating on the inside of the pipe. Hardness is also expressed as the equivalent quantity of $CaCO_3$ (calcium carbonate).

Thomas et al., 1981). Other properties of the water, including temperature (e.g., Mullen and Ritter, 1980; Britton and Richards, 1981); velocity; treatment with chlorination or chloramination* (Treweek et al., 1985); presence of humic substances (Moore, 1973; Samuels and Meranger, 1984); chloride and nitrate levels (Oliphant, 1982); and dissolved oxygen or other elements, may also affect plumbosolvency.

II.A.1.b. Lead Solder

The use of lead/tin solder, in a tin to lead ratio of 50:50 or 60:40, is ubiquitous in U.S. residential plumbing at present (e.g., Lead Industries Association, 1982; Chin and Karalekas, 1984; AWWA-DVGW Report, 1985). Lead solder is probably the greatest single contributor to lead contamination of drinking water in this country because of its widespread use and easy volubility. Its easy volubility is caused by the galvanic reaction between the lead/tin solder and the copper pipes that are used most commonly in residential plumbing (Anderson, 1984). Many recent studies have shown that solder containing lead, when used with copper household plumbing, could easily raise lead levels above 50 ug/l, even when in contact with relatively non-corrosive water or within a relatively short period of time (e.g., Wong and Berrang, 1976; Nielsen, 1976; Department of the Environment, 1977; Lyon and Lenihan, 1977; Lovell et al., 1978; Britton and Richards, 1981; Sharrett et al., 1982a and b; Oliphant, 1982 and

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* Chemicals used in drinking water disinfection.

1983; Samuels and Meranger, 1984; Birden et al., 1985; Treweek et al., 1985). Some of these and other investigations (e.g., Department of the Environment, 1977) also found that lead solder alone could produce lead contamination levels that were as high or higher than those in wholly lead-plumbed houses. Indeed, under some conditions, because of the galvanic action they can be much higher (Oliphant, 1982 and 1983; Murrell, 1985). Data summarized in the AWWA-DVGW Report (1985), Table 4-19 and elsewhere, show that even without new solder, the galvanic reaction in relatively non-corrosive waters (pH 7.5-8.5, alkalinity > 100 mg/l as $CaCO_3$) can produce lead levels at the tap of 160-250 ug/l upon overnight standing. Because both the solder and the copper piping must be exposed, however, galvanic corrosion is usually a more serious problem with new plumbing.

Many studies have shown that the age of the lead solder is among the most important variables affecting volubility. For example, Sharrett et al. (1982a) -- studying Seattle, a city with few lead pipes -- found that the age of the house (a proxy measure for the age of the solder and other plumbing materials) was the dominant factor for predicting the concentration of lead in the tap water. In homes that were newer than five years old, with copper pipes, the median lead concentration for standing water was 31 ug/l versus 4.4 ug/l in older homes. In homes built within the previous 18 months, the median lead level was 74 ug/l.

New solder will leach lead even in relatively non-corrosive water -- whether naturally less corrosive or treated (e.g., Nielsen,

1976; Herrera et al., 1981) -- and will continue to leach significant amounts for up to five years (Lovell et al., 1978, Sharrett et al., 1982a; Oliphant, 1982 and 1983; Murrell, 1984; Lassovszky, 1985; Neff and Schock, 1985; etc.) . Murrell (1985) found that new solder could leach sufficient lead to contaminate standing water to a degree hundreds of times higher than the current MCL; this has been confirmed by data collected by the Philadelphia water utility (1985) and elsewhere. Oliphant (1983) has also shown that no matter how small the area of exposed solder, provided the contact time is long enough, the lead levels will always exceed 100 ug/l if the volume of the sample is small.

With new (exposed) solder, the duration of contact need not be long to raise lead levels significantly. Britton and Richards (1981) and Lyon and Lenihan (1977) have shown that, with particularly corrosive drinking water, lead levels in standing water in systems with copper plumbing joined with lead solder could rise above 100 ug/l within 40 minutes of contact. Oliphant (1983) has presented evidence that those conditions can produce lead levels one to two orders-of-magnitude higher than expected from equilibrium volubility calculations.

Two other factors will affect the rate of lead leaching from lead-soldered joints: the surface area of the lead/tin solder at the joints (which can often relate to the quality of the plumbing and jointing work) and the number of joints per length of pipe (e.g., Nielsen, 1975 and 1976; Lyon and Lenihan, 1977; Walker and Oliphant, 1982b; Oliphant, 1983; Birden et al., 1985; Lassovszky,

1985; Murrell, 1985; Treweek et al., 1985). It has also been suggested that with pipes running vertically, the lead solder can drip down the pipe, resulting in more exposed solder than in horizontal pipes (Arthur Perler, U.S. EPA, private communication).

Lead solder in brass kitchen faucets can result in particularly high concentrations of lead (Samuels and Meranger, 1984).

Only one paper that we are aware of (Thompson and Sosnin, 1985) suggests that lead solder does not contribute significant amounts of lead to water. In that article, although the measurements are presented as first-draw standing levels after a 12-hour exposure period, the data shown are really for flushed (running) samples (termed 'steady-state values'). The article was based on a report (Battelle, 1982) that contains the actual data on lead levels. In that report, first-flush samples averaged above 50 ug/l (Figures 22, and 23, and Table 50 of the Battelle report). This is acknowledged in the narrative on page 33 of the report, but discounted because the lead content decreases with time. Therefore, the results of the Battelle study are consistent with the rest of the literature.

The easy plumbosolvency of lead solder has been known for many years. As a result, the Netherlands banned lead solder in 1977, Germany banned the use of lead solder about the same time, and several states and localities in the United States have banned it within the past few years. The 1986 Amendments to the Safe Drinking Water Act prohibit the use of materials containing lead in public water systems, a prohibition that is enforceable by the States after June 1988.

II.A.1.c. Lead Pipes

Several characteristics of lead piping also influence lead levels in drinking water (Pocock, 1980; Britton and Richards, 1981; etc.). The length of the lead pipe, in both the home and the supply lines, can have a positive association with lead levels Kuch and Wagner, 1983; Department of the Environment, 1977; Pocock, 1980; Karalekas, 1984) as can the position of the pipe -- it appears that, even for the same length of pipe, water composition, etc., lead piping contained wholly or partly within the house (as opposed to lead service connections outside the house) correlates with higher first-draw lead levels. The ratio of the surface area of lead exposed to the water volume contained is also important (Ainsworth et al., 1977). The age of the dwelling or the pipe (Ainsworth et al., 1977) and the percentage of lead piping in both the service mains and within the residence are significant in determining lead levels, as well. New lead pipes appear to leach higher levels of lead initially, with the rate decreasing within a few days or weeks (Ainsworth et al., 1977).

II.A.1.d. Other Potential Sources of Lead

Lyon and Lenihan (1977) and others have found that the flux used for soldering is an excellent electrolyte and can contribute significant amounts of lead to drinking water.

Some lead can also leach from copper pipes themselves (Herrera et al., 1981). Specifications for copper pipes usually limit only copper and phosphorus, and copper used for non-drinking water applications is permitted to contain some lead. Copper pipe manufacturers have indicated that copper tubing used for

water can be made from recycled copper products, thus permitting the introduction of lead impurities (Herrera et al., 1981). Although not common, lead impurities can also occur in galvanized pipe (e.g., Nielsen, 1975).

Lead is also used in the production of brass and bronze. Brass is a copper-zinc alloy, which can contain up to 12 percent lead, and bronze is a copper-tin alloy, which can contain up to 15 percent lead (U.S. EPA, 1982b). While both are relatively corrosion resistant and are not generally recognized as a source of lead, several studies document lead leaching from brass or bronze fixtures (Nielsen, 1975; Samuels and Meranger, 1984; Neff 1984; Neff and Schock, 1985; Neff et al., 1987).

Lead can also contaminate potable water when used in pipe jointing compounds and through its use for goosenecks, valves, and gaskets in water treatment plants or distribution mains.

At least one study (Herrera et al., 1982) found that lead can leach from tin-antimony solder “presumably com[ing] from impurities in the solder.”

Early tests of plastic pipes showed that lead contamination resulting from stabilizers used with polyvinyl chloride (PVC) pipes could be high (studies summarized in National Academy of Sciences, 1982; volume 4, pages 64ff). However, since then, the National Sanitation Foundation has developed a standardized testing procedure for plastic pipes.

Additional analyses of the leaching of lead from these and other sources is needed (AWWA-DVGW Report, 1985).

II.A.1.e. Other Factors Relating to Lead Contamination Levels

The most important other variable affecting lead levels in drinking water is the length of contact time between the water and the plumbing materials. High lead levels are found in water from faucets that are seldom used or in the first drawn samples in the morning after the water has sat overnight. Usually, flushing the faucet will significantly decrease the lead levels in the water. This is true for all waters, corrosive and less corrosive. With very corrosive waters or new solder, the length of contact time need not be great -- as little as 40 minutes to an hour can produce lead levels above 100 ug/l under certain conditions (e.g., Lyon and Lenihan, 1977; Britton and Richards, 1980; Kuch and Wagner, 1983; Oliphant, 1983).

The number of occupants of the dwelling is inversely proportional to lead levels, probably because fewer occupants means the water will, on average, remain in the pipes longer (Department of the Environment, 1977; Pocock, 1980).

Other factors that can affect lead levels in tap water are pipe length and diameter, and water velocity (Bailey and Russell, 1981; Kuch and Wagner, 1983). No matter what metal the pipe is made of, the diameter of the pipes is inversely proportional to lead contamination levels because of the greater proportion of water in contact with the lead-containing surface (solder, flux or pipe) in pipes with smaller diameters (Crank, 1975). The length of the metal pipe can correlate directly with the potential for high lead concentrations although not consistently (Sharrett

et al., 1982a; Kuch and Wagner, 1983). Lead levels can also increase with the turbulence and velocity of the water, and with irregular flow patterns.

II.A.1.f. Summary

Because lead levels in tap water are a complicated function of the specific qualities of the water, the particular materials comprising the distribution and household plumbing, and the length of time the water is in contact with the plumbing materials as well as other factors, there is a high degree of within-house and between-house variability in water lead levels (Sartor et al., 1981; Bailey and Russell, 1981). With even mildly aggressive (corrosive) water, however, any amount of lead anywhere in the distribution system or household will contribute some lead to the drinking water. Overall, there are four major risk factors: the the degree of corrosivity, the length of time in the pipe, the total amount of lead in the plumbing materials, and the newness of the plumbing.

In general, lead levels in first-draw water can be several times higher than in running water (e.g., Battelle, 1982). With aggressive waters and new solder, however, first-draw samples can easily be an order-of-magnitude or more greater than running levels (cf. data in Karalekas et al., 1975; Oliphant, 1983; Maessen et al., 1985; Murrell, 1985; etc.). Lead concentrations in fully flushed samples typical of distribution system water,

even under corrosive conditions and with new solder, are generally below 50 ug/l and are usually below 20 ug/l.*

II.A.2. Prevalence of Lead Materials in Distribution Systems

Lead has been used for plumbing materials since at least Roman times. It was considered a convenient and suitable conveyance of water and was used extensively for water pipes during the nineteenth and early twentieth centuries.

The danger of lead contamination of drinking water was not unknown, however. In 1845, the Report of the Commissioners to Examine the Sources from Which a Supply of Pure Water May Be Obtained for the City of Boston concluded, "Considering the deadly nature of lead poison, and the fact that so many natural waters dissolve this metal, it is certainly [in] the cause of safety to avoid, as far as possible, the use of lead pipe for carrying water which is to be used for drinking." Lead pipes were outlawed in several German states in the second half of the nineteenth century because of concern over health (cited in AWWA-DVGW Report, 1985; p. 223). And a warning of potential danger from lead pipes was given to the New England Water Works Association in 1900 (Forbes), showing that the risk was known there, as well.

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* This pattern of lead contamination also holds in Canada. Data on lead contamination in raw, treated and distributed water from 70 municipalities across Canada show levels averaging 1 ug/l (Meranger et al., 1979). On the other hand, a study of lead levels in waters that had sat overnight in home plumbing showed 20 percent of the samples exceeding 50 ug/l, with a mean level of 43 ug/l (Maessen et al., 1985).

Despite these and other warnings, in 1924 Donaldson reported that half of the 539 cities he surveyed in the early 1920s used lead or lead-lined service pipes. He found the greatest usage of lead service lines in New England, New York, the Midwest, Texas, Oklahoma and Montana.

Because lead is a relatively durable material, many of the original lead lines are still in service (Patterson and O'Brien, 1979).

More recently, Chin and Karalekas (1984) surveyed 153 public water systems in 41 states, Puerto Rico and the District of Columbia, to ascertain the prevalence of lead materials in distribution systems. Their survey targeted large systems, with 91 of the 153 systems surveyed serving populations over 100,000 people. (Nationally, only 0.5 percent of community water systems serve over 100,000 people.) They found that almost three-quarters of the systems had used lead service lines or connections (most of the remaining quarter did not know if they had any lead or lead-lined services), and one city (Chicago) still installed lead service lines.* In addition, almost two-thirds of the systems had lead goosenecks in their plumbing (another 10 percent didn't know if they had any) and about half of the systems reported the use of solder or flux containing lead in the distribution system.**

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* The installation of new lead pipes is now prohibited in Chicago.

** The utilities may be referring to use in home plumbing or in service lines.

The use of lead/tin solder is ubiquitous in U.S. residential plumbing at present. Indeed, until 1986, most local plumbing and construction codes recommended or even required the use of copper pipe joined by lead/tin solder. The 1986 Amendments to the Safe Drinking Water Act banned the future use of materials containing lead in public water systems or in residences connected to public systems. The ban became effective immediately (June 19, 1986), but States have up to two years to enforce this provision.

II.B. Data on the Occurrence of Lead in Drinking Water

Under the provisions of the Safe Drinking Water Act, EPA must ensure that public drinking water supplies are free of contamination and that they comply with primary drinking water regulations; this authority includes setting monitoring requirements to assess compliance. Sections 1401(1)(D) and 1445(A)(1). According to EPA regulations, monitoring for inorganic compounds, including lead, must be conducted once per year for systems whose source is surface water and once every three years for water supplies using ground water. 40 CFR §141.23. MCLs are defined as "the maximum permissible level of a contaminant in water which is delivered to the free flowing outlet of the ultimate user of a public water system." 40 CFR § 141.1(a). "Free flowing" has been generally understood by the States and the water utilities to mean a "fully flushed" sample. In addition, the procedures for laboratories certified for reporting purposes under EPA's Laboratory Certification Program, administered by the Office of Drinking Water, specify that the sample be taken after running the water for two

or three minutes (U.S. EPA, 1982c). However, because lead is a corrosion by-product, these procedures decrease the likelihood of detecting lead contamination. Therefore, the data collected for compliance with the Safe Drinking Water Act, as administered currently by EPA, does not adequately represent exposure to lead in U.S. drinking water.

Several studies have investigated the quality of drinking water in the United States, including lead levels (e.g., the National Inorganic and Radionuclides Survey, the National Organics Monitoring Survey). But these surveys have also not addressed the phenomenon of most lead contamination of drinking water -- as a corrosion by-product. These surveys have sampled lead levels in fully-flushed water typical of distribution water. Again, this sampling procedure minimizes the likelihood of detecting the contamination of tap water by lead. Several other studies of national water quality have been conducted over the past two decades (e.g., the 1969 and 1978 Community Water System Surveys, the Rural Water Survey, the First National Health and Nutrition Examination Survey), but the results from those surveys have not been used by EPA in setting national standards. (See, for instance, U.S. EPA, 1985; or 40 CFR Part 141, page 46969.)

The estimate of occurrence, therefore, is based upon data collected and analyzed for EPA's Office of Drinking Water in 1979-81. These data portray partly-flushed (30 seconds) kitchen tap samples collected by the Culligan Water-Softening Company;*

* The use of company names and the presentation of related data does not constitute endorsement of their services.

James Patterson of the Illinois Institute of Technology analyzed the data. EPA believes that the Culligan tap samples are more representative of consumed water than are the fully-flushed samples taken in compliance with EPA's monitoring regulations. In addition, these samples of partly-flushed, random daytime water are more appropriately used with the water-lead-to-blood-lead equation accepted by EPA than would be data on fully flushed water or even a value integrating average consumption patterns.

After the presentation of the Culligan data, this section also includes a discussion of potential biases in the data, the consistency of the findings with field and laboratory results, and an alternative analysis of the potential contamination of drinking water by lead (to confirm the magnitude of the estimates).

Finally, because homes with newly-installed plumbing containing lead solder or flux have a higher risk of elevated lead levels than homes with older plumbing, this section also discusses exposure to lead likely to occur in new housing.

II.B.1. Patterson Study/Culligan Data on Tap Water

In 1979-1980, EPA's Office of Drinking Water funded a study by James Patterson using data on residential water quality (Patterson, 1981). The study, "Corrosion in Water Distribution Systems", analyzed 772 municipal water samples collected by Culligan Water Softening dealerships in 580 cities in 47 states. The samples were collected from May to November 1978 at the consumers' kitchen taps. No samples were collected from households using home water softeners.

The purpose of this study was to evaluate the relationship between different corrosion indices and water quality variables likely to influence water corrosivity against the observed levels of the corrosion by-products iron, copper, lead and zinc. The impetus for EPA's commissioning of this analysis was probably the suggestion of the court in a lawsuit brought by the Environmental Defense Fund against the Agency. EDF v. Costle, 578 F.2d 337, 349-50 (D.C. Cir. 1978). In addition to containing data on the levels of these metals, the analysis contained information on calcium, magnesium, sodium, pH, alkalinity, chloride, conductivity, sulfate, and silica levels in each sample. Calculations of hardness and corrosivity indices (including Langelier, Ryznar, Aggressive, Driving Force, and Larsons, as well as Dye's Momentary Excess) are also presented.

Water samples were collected by Culligan Dealership representatives throughout the United States, after the kitchen tap had been flushed for 30 seconds at a moderate flow rate, according to a standardized sampling procedure. All samples were collected in virgin plastic polypropylene bottles with plastic screw tops. The metals analyses were conducted by the Illinois Institute of Technology and the other analyses were done by the Culligan laboratory in Northbrook, Illinois. Most samples were collected between 10:00 a.m. and 8:00 p.m.

For lead determinations, lithium nitrate was added to the acidified sample (although not until the samples were transferred to glass bottles) and the flameless graphite furnace atomic

absorption procedure was employed. The standard addition technique was used for all lead determinations, to avoid certain common interference problems. Standard Methods (1971) were employed for the analysis of all samples, except for the atomic absorption procedures, where Perkin-Elmer (1977) procedures were used.

Analytical tests were conducted to evaluate potential testing and analytical biases, both upward and downward. To test for upward bias (contamination that would increase apparent lead levels), nitric acid was stored in the plastic sample bottles for two weeks to draw out any lead in the plastic bottle itself, which could otherwise contaminate the sample. The liquid was then tested for lead presence, and there was none. Blank samples (i.e., bottles filled with lead-free distilled water) were also used to check for upward bias. After a period of standing (two weeks), the distilled water was tested for lead contamination; it remained lead-free. These efforts showed that contamination of the samples from the plastic bottle itself was unlikely.

On the other hand, because no nitric acid was used to preserve the samples until the water samples were transferred to glass vials, there was a possibility that some lead from the sample would adsorb to the plastic bottle; this would bias the results downward (i.e., lower the measurable lead level). Spiked samples (i.e., solutions with a known amount of lead) were used to test for this. The results indicated that, on average, 3 ug/l of lead adsorbed onto the plastic bottle; therefore, a slight downward bias was present in the results.

Calibration and other analytical controls were also employed at several points in each batch as part of the laboratories' ongoing quality assurance and quality control efforts. (Full documentation is available from each lab.)

These data indicate that 16 percent of partly flushed water samples could exceed an MCL of 20 ug/l at the kitchen tap, and that 3 percent exceed the current MCL of 50 ug/l. Fifty-two percent of the samples contained 9 ug/l or less of lead, the maximum was 10,000 ug/l. The occurrence of high lead levels was geographically widespread. Samples with lead levels greater than 20 ug/l were taken in more than half of the states in the country, and in every one of EPA's ten Regions. The distribution of lead levels in these samples is presented in Table II-1.

II.B.1.a. Consistency with Other Data

The literature on contamination levels due to lead leaching from plumbing materials shows great variations relating to the specific conditions being observed.

The Culligan data, with 16 percent of the samples exceeding 20 ug/l, have a lower incidence rate of high lead concentrations than is commonly portrayed in the literature on lead leaching rates and the potential for lead contamination in tap water. This is reasonable because that literature, in general, focuses upon high risk (i.e., very corrosive) waters.

TABLE II-1. Distribution of Culligan Data (Patterson, 1981)

	Measured Lead Concentrations (ug/l)			
	< 10	11-19	20-49	≥ 50
Percent of samples	60	24	13	3

The Culligan samples, on the other hand, do not portray particularly corrosive waters. In these samples, the median pH is 7.2 (mean, 6.8), median alkalinity is 106 mg/l as $CaCO_3$ (mean, 144 mg/l), and median hardness is 145 mg/l as $CaCO_3$ (mean, 203 mg/l). These are considered to be relatively "non-corrosive" waters. Comparing the hardness of the water in these tap samples, for instance, with the U.S. Geological Survey estimates of the extent of soft water in the United States (Durfor and Becker, 1964a and 1964b) or the data in Millette et al. (1980),* these samples represent water that is much harder than the average in the country. The average Langelier Saturation Index** for the Culligan samples is -0.4, which is fairly stable.† It is logical that less corrosive waters would contain a lower incidence of corrosion by-products than do studies of more corrosive water.††

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* Both of these studies are discussed more extensively below in Section B.1.C. and in Chapter V.

** One index used to estimate a water's potential corrosivity, discussed more fully in Chapter V.

† A "stable" water is one where a film of $CaCO_3$ should be exactly at equilibrium, i.e., it neither dissolves nor deposits and grows.

†† While it is hard to generalize about these studies, typical results under corrosive conditions show from perhaps 50-100 percent of the samples exceeding 50 ug/l (e.g., Lyon and Lenihan, 1977; Department of the Environment, 1977; Britton and Richards, 1980; Oliphant, 1983) to 15-50 percent exceeding 50 ug/l (e.g., Karalekas et al., 1977; Karalekas et al., 1978; Seattle Water Metals Survey, 1978; Craun and McCabe, 1975). By comparison, the Culligan estimate (16 percent >20 ug/l) is low.

The Culligan data portray higher levels of lead contamination, however, than do either. EPA's data on compliance with the Safe Drinking Water Act or most of the studies that have investigated the quality of distributed drinking water in the United States, including lead levels. These surveys and EPA's monitoring requirements have measured lead levels in water more typical of the distribution system and most of the results indicate low levels of lead contamination.* Lead contamination, however, occurs primarily in tap water and in water that has been in contact (standing) with pipes for some length of time. Therefore, the somewhat higher lead levels in the partially flushed, random daytime tap samples that make up the Culligan samples confirm the findings from experimental and field studies of lead leaching rates and patterns.

Perhaps the clearest indication of the consistency of the Culligan samples with other data on lead contamination of drinking water is a comparison with the preliminary results of EPA's "Lead Solder Aging Study", presented in U.S. EPA (1987). The lead levels in the partially flushed Culligan samples are similar to those in housing over two years old with median pH (i.e., 7.0-7.4) or in waters with high pH (i.e., > 8.0). The data from the Lead Solder Study are presented in Tables II-3 and II-4, below.

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* For comparison, the National Inorganic and Radionuclides Survey, recently conducted by EPA, shows only about 1.5 percent of ground-water-supplied public systems have lead levels over 20 ug/l in fully flushed water.

The Culligan data serve as the basis for this analysis because, of the available data, EPA believes that they are more representative of the water consumed in this country than are the fully-flushed samples taken in compliance with EPA's monitoring regulations. People drink the water from the taps in their homes after it has been sitting for unknown periods of time. While some people may run their water before using it for cooking or drinking, probably many people (especially children) do not. These data portray neither an upper bound for exposure (which would result from analyses of first-flush samples) nor a lower bound (which would come from an analysis of fully-flushed distribution water); they more closely portray the bulk of the water that is consumed (e.g., Bailey and Russell, 1981). Finally, when controlling for other environmental sources of lead exposure, studies of the relationship between blood lead levels and water lead have also found a better fit with lead levels in standing water than with fully-flushed samples (e.g., Worth et al., 1981; Bailey and Russell, 1981; Pocock et al., 1983).*

II.B.1.b. Potential Biases in the Data

In determining whether it is reasonable to generalize from a subset, it is important to determine any potential biases in the data and, if found, to assess the likely effect upon the results. Some potential biases (for example, potential analytical and

* This issue is presented more fully in the section below on uncertainties, within the discussion of the relationship between blood lead levels and levels of lead in drinking water.

testing biases) as well as the consistency of the findings with the literature on water quality and lead leaching rates were discussed in previous sections. Others, for instance, the inclusion of new housing within the data, are discussed below. In this section, we address the issues of potential selection bias, geographic representation, and the implications of the relative hardness of the samples.

Because these samples were collected by a water treatment company, a self-selection bias is possible; i.e., it is conceivable that they represent water that is 'dirtier' than average. However, Culligan is not a general water treatment company and does not test for contamination by inorganic (e.g., lead) or organic (e.g., pesticides) substances. EPA and the Illinois Institute of Technology arranged for and conducted the analyses of metal contamination in these samples. Homeowners who had general water problems or who had reason to suspect that their water was contaminated would be unlikely to call Culligan for water testing or treatment. If they did call and outline such a problem, the Culligan representative would have either suggested that they contact another lab in the area or informed them that Culligan would charge extra to arrange for such analyses. Therefore, it is unlikely that there would be selection bias resulting in higher lead contamination levels.

On the other hand, a different selection bias is likely and did, in fact, occur. Not surprisingly, because the samples were taken by a water softening company, overall hardness results from

the Culligan/Patterson data were higher than other data on water hardness in the country.* For comparison, only about 10 percent of the Culligan samples contained soft water (i.e., < 60 mg/l as $CaCO_3$) while in the U.S. Geological Survey (Durfor and Becker, 1964a and b),** about one-third of the country had soft water. National patterns portrayed in the U.S. Geological Survey held in this survey: the Northeast and Southeast had the softest water, with the Midwest averaging almost three times harder water. The Northwest also had relatively soft water.

The highest lead levels in this data set were in the Midwest, which also had the hardest water. The combination of high hardness and high lead levels is somewhat surprising; usually lead (as a corrosion by-product) occurs more frequently in softer water. Schock (1980 and 1981) and Schock and Gardels (1983) have shown, however, that at a pH of 8-8.5, soft water (i.e., 30 mg/l as $CaCO_3$) may be less corrosive to both lead and copper than can be hard water (i.e., > 150 mg/l). These results confirm Patterson's findings (1981) and the consensus of the technical literature: Many inter-related factors affect a water's corrosivity and no single corrosivity index adequately measures the actual corrosion potential of a specific water; therefore, no single index is a good predictor of corrosion by-products, including lead.

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* The range in hardness in the Culligan data is extreme: from 2 to 975 mg/l as $CaCO_3$, with median 145 and mean 203. Generally, levels over 240 mg/l are quite uncommon.

** The USGS data is discussed more fully in Chapter V.

But while no single measure of water quality is a good predictor of the actual corrosion potential of a specific water, the weight of the literature does show that softer water tends to be more corrosive than harder water. Therefore, this selection bias may introduce some downward bias to the estimates; i.e., the concentrations of lead in the Culligan data may be somewhat low relative to the actual levels in the country as a whole.

Because the people who call Culligan can afford to pay for those services, another potential self-selection bias is that the population represented by these data is wealthier (and possibly more sensitive to health and environmental issuses) and contains a greater proportion of single-family and owner-occupied housing than is typical in the U.S. population. It is unclear what effect this could have on the estimates. A brief discussion of possible contamination patterns in multi-family housing, particularly high-rise buildings, is included in the section on uncertainties, below.

A question arises as to whether the samples are geographically representative of the United States. The percentage of samples collected from each state closely reflects the population distribution across the United States, with a few exceptions. As a proportion of percentage-of-samples to percentage-of-U.S.-population, the most significant differences are the states of Alabama, Arkansas, North Carolina and Washington, where the difference varies by about an order of magnitude. For the state of Wyoming, the difference is a factor of 5. These states are

relatively small and combined, contain only about 7 percent of the U.S. population. This could be expected because the smaller the size of the observation, the larger the relative effect. The anticipated effect on the estimate is low. For an impact upon the final estimates, the most significant differences are the states of California, Colorado and Illinois, where the absolute difference between the percentage of samples and percentage of population differs by more than 5 percent of the total. On a regional basis, however, the distribution of samples closely paralleled the population distribution. The most significant exception here is the Midwest; the percentage of samples (42 percent) was much larger than the population (26 percent, 1980 Census). Table II-2 presents the distribution of samples by state, and by region of the country.

We conducted chi-square tests* of the distributions to determine how closely related they were. The results were inconclusive, however, because whether the results were significant depended upon how finely subset the data were, that is, how many divisions (and, therefore, how many degrees of freedom) for the data were used. The possibilities included 50 subsets (i.e., by state, with 49 degrees of freedom), 10 (i.e., by EPA Region, with 9 degrees of freedom), 5 (i.e., using Patterson's groupings, with 4 degrees of freedom), or simply for the nation as a whole.

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* The chi-square statistic, represented by the Greek letter x raised to the 2nd power, is a measure of how much, proportionally, the frequencies in the observations differ from the frequencies you would "expect" if there were absolutely no relationship between the variables (Matlack, 1980).

TABLE II-2. Municipal Water Samples and Population Percentages from Culligan/Patterson Study (Patterson, 1981)

State	% Population*	% Samples
Northeast States		
CT	1.4	0.63
MA	2.5	2.4
ME	0.4	0
NH	0.4	0.38
NJ	3.3	5.2
NY	7.8	5.3
PA	5.3	3.9
RI	0.4	0.50
VT	0.2	0.25
Total	22%	19%
Southeast States		
DE	0.3	0.25
FL	4.3	2.9
GA	2.4	1.8
MD	1.9	1.3
NC	2.6	0.25
SC	1.4	0.63
VA	2.4	2.6
WV	0.9	2.4
Total	16%	13%
Midwest States		
IA	1.3	3.7
IN	2.4	6.2
IL	5.1	12.4
KS	1.0	1.9
MI	4.1	2.0
MN	1.8	4.2
MO	2.2	2.3
ND	0.3	0.38
NE	0.7	1.3
OH	4.8	4.6
SD	0.3	0.76
WI	2.1	2.0
Total	26%	42%

* U.S. Bureau of Census, 1980. In the Patterson study, 1978 Census data were used.

TABLE II-2. (Continued)

State	% Population*	% Samples
Southcentral States		
AL	1.7	0.25
AR	1.0	0
KY	1.6	1.0
LA	1.9	1.3
MS	1.1	0.63
OK	1.3	0.88
TN	2.0	1.8
TX	6.3	5.2
Total	17%	11%
Western States		
AZ	1.2	2.0
CA	10.5	2.3
CO	1.3	6.3
ID	0.4	0.51
MT	0.3	0.88
NM	0.6	0.88
NV	0.4	0.51
OR	1.2	1.3
UT	0.6	0.25
WA	1.8	0.13
WY	0.2	1.0
Total	19%	16%

* Bureau of Census, 1980

To check whether the slight skewing of number-of-samples compared to population-by-state affected the estimates, we weighted the results by state population and compared that analysis with the results of the national analysis (which assumed that the data are geographically representative of the nation). The results differed by less than one-half of one percent. We concluded that the slight variation in geographic distribution does not significantly affect the estimates.

II.B.1.c. Alternative Analysis of Potential Exposure to Lead in Drinking Water

Several bodies of literature are available for an alternative analysis of potential exposure to confirm the magnitude of the results from the Culligan data. These include studies of the extent of highly corrosive water in the United States and experimental and field analyses of lead contamination.

Two major studies have focused on assessing the extent of highly corrosive water in the United States.* They are the U.S. Geological Surveys (USGS) conducted in the early 1960s (published in Durfor and Becker, 1964a and 1964b) and the First Health and Nutrition Examination Survey (HANES I), conducted by the National Center for Health Statistics in 1974 and 1975.** The results of

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* This information is presented more fully in Chapter V, section B.1.

** The data from the Midwest Research Institute (1979) and from Millette et al. (1980) on the extent of corrosive water in the country contain profiles that are quite similar to the USGS and the HANES I. They are not included in this discussion, however, because this analysis addresses estimates of the extent of specifically soft water, not water that is corrosive for other reasons. Chapter V discusses these two studies as well.

these studies can be combined with analyses of lead leaching rates and the variables affecting plumbosolvency for comparison with the findings from the Culligan data.

The USGS data showed that 17 states had very soft water (under 60 mg/l as $CaCO_3$); using 1980 Census data, the combined populations of those states is 67.7 million people. The results of the HANES I (as published in Greathouse and Osborne, 1980) show a very similar picture: about one-third of the country has very soft water (under 60 mg/l as $CaCO_3$). Given a total current (1985) national population of a little over 240 million, about 80 million people receive very soft water.

Numerous studies of plumbosolvency conducted in the United States and in Great Britain have shown that soft, acidic waters are most corrosive and have the highest lead contamination levels (e.g., Craun and McCabe, 1975; Nielsen, 1976; Hoyt et al., 1979; Patterson and O'Brien, 1979; U.S. EPA, 1982b; Sheiham and Jackson, 1981; Worth et al., 1981). There is some discussion as to whether pH or carbonate content is the most important variable, and many studies show that the relationship between pH or carbonate and lead levels may be neither simple or linear. In general, the lower either value is, the more vulnerable the water to high lead contamination.*

Both laboratory and field experiments demonstrate this. For instance, data from Karalekas et al. (1977) on two cities

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* Because neither the relationship between pH nor carbonate and lead is linear, this is not strictly true at all pH or carbonate levels. Kuch and Wagner (1983) and Schock and Gardels (1983) among others have developed multi-dimensional models of lead solubility.

(Bridgeport, Connecticut, and New Bedford, Massachusetts) with similar pH levels (7.1 and 7.3, respectively) but different hardness and alkalinity (48 and 18 mg/l vs. 12 and 24 mg/l, respectively) indicated that even with moderate pH the softer water could contain six to eight times higher lead contamination levels. Schock and Gardels (1983), investigating water with high pH (> 8.6) but low carbonate content, present average lead concentrations of 67-134 ug/l in first draw water. Other results typical of this literature are the field data presented in the Seattle Water Metals Survey (1978) and the laboratory data in Sheiham and Jackson (1981). In the former, highly corrosive waters produced mean lead levels of almost 30 ug/l (from lead solder alone; there were no lead pipes) and in the latter, mean lead levels were over 100 ug/l with a mixture of lead and non-lead pipes (some old, some new) and lead solder. In sum, most studies show that first flush samples from soft, corrosive water (i.e., hardness under 60 mg/l as $CaCO_3$) often result in lead contamination levels exceeding 50 ug/l* even in housing that is not new. (New housing is at particular risk of high lead levels. This is discussed in the next section.)

Not everyone who receives very soft water, however, is at equal risk of high lead exposure. Some water systems with very corrosive waters (Boston and Seattle, for instance) treat their water to reduce its aggressiveness; their risk is lower. A small proportion of water utilities have lead in source water, as well

* Because EPA's standard for lead is 50 ug/l, most studies have used that as the cut-off.

as a corrosion by-product; their risk is higher. Some localities have few lead pipes and connections (and hence, have a smaller likelihood of high lead contamination), while others have many. Some localities have begun already to ban the use of materials containing lead in public water supplies and in residences connected to them. In addition, plastic pipes are beginning to be used in some residential plumbing, even for bringing in potable water,* replacing the metal pipes that are more likely to leach lead. Finally, at least one city (Akron, Ohio) has instituted an active program to replace its lead service connections.

To account for these variables and for the idiosyncrasies of specific waters, we assumed conservatively that with very soft waters (i.e., hardness under 60 mg/l as $CaCO_3$), half of first-flush samples could exceed 20 ug/l, and that 10 percent of them could exceed 50 ug/l.

Combining information, then, on 1) the extent of very corrosive water in the country (about one-third of the population receives very soft water) and 2) studies of potential lead contamination with very soft water (almost all have levels > 20 ug/l in first-flush samples and many also had levels > 20 ug/l in random daytime samples) but 3) mitigated to include some factors likely to decrease exposure* (e.g., some cities have already begun corrosion control treatment) yields the following calculation:

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* Plastic pipes are used more commonly for waste water than for intake.

1/3 of country x 50% occurrence of Pb>20 ug/l =
17% of population exposed to water >20 ug/l

Therefore, this alternative estimate of potential exposure to lead contamination of drinking water in non-new housing yields results that are quite close to those of the Culligan data analyzed by Patterson (1981), where 16 percent of the samples were greater than 20 ug/l. The results are most sensitive to the estimate of lead levels > 20 ug/l. (For comparison, assuming 25% occurrence yields a national estimate of 8% and assuming 75% occurrence yields a national estimate of 25% of the U.S. population exposed to water > 20 ug/l.)

There is additional anecdotal evidence that also supports the patterns and extent of lead contamination presented here. A CBS-affiliated television station in Cleveland, Ohio (Channel 8) conducted a small survey of lead levels in first-flush tap water in early 1987. They found 13 percent of the samples exceeded 20 ug/l, with a home occupied for only three months having lead levels of $\geq$ 100 ug/l. Second, a water utility in Colorado (Little Thompson Water District) conducted a limited survey of lead contamination within customers' residences. This survey was conducted in conjunction with the Colorado Department of Health and was accomplished concurrently with sampling programs in the cities of Denver, Colorado Springs, and Fort Collins (Colorado). The results from Little Thompson showed one-third of the residences sampled exceeded 20 ug/l -- results they indicated "were comparable

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* Some factors, for instance, many lead pipes or lead contamination of distribution water, can increase exposure estimates, also.

to the results of the aforementioned cities." The highest reading (> 400 ug/l) was in a not-yet-occupied house. Third, concern about potential lead contamination of drinking water in Washington, D.C. in late 1986 and early 1987 resulted in about 1,000 water samples being taken there. Newspaper reports (Washington Post) and numerous press releases from the D.C. Department of Public Health reported that between 13 and 25 percent of the water samples exceeded 20 ug/l, and that the results indicated “that as many as 56,000 houses may have problems with lead contamination," resulting from the common use of long lead service connections in many parts of the city. In addition, data collected by KYW-TV in Philadelphia (an NBS-affiliate) in February 1987, by EPA’s Region IX (San Francisco) in Spring 1987, by the Nassau County (New York) Department of Health in 1987, and jointly by the New Jersey Department of Environmental Protection and the U.S. Geological Survey (presented by J. L. Barringer at the American Geophysical Union Spring 1987 meeting) show widespread lead contamination following predictable patterns: contamination levels are high with new plumbing or corrosive waters, contamination is minimal with non-corrosive waters and older plumbing.

Finally, data available from WaterTest Corporation,* a private water testing laboratory in Manchester, New Hampshire, on over 2,500 samples taken in January-March, 1987, show that average first-flush samples exceeded 20 ug/l in 15 states plus the District of Columbia. This shows that the occurrence of lead levels exceeding 20 ug/l is widespread in this country but it was not

* The use of company names and the presentation of related data does not constitute endorsement of their services.

possible to determine the age of the housing from which the samples came.

This alternative analysis of potential lead contamination of tap water was based upon assumptions about leaching rates in soft water (< 60 mg/l as $CaCO_3$), but this is not the only factor that makes water corrosive. Many other parameters, including pH, alkalinity, temperature, etc., contribute to corrosivity.

People who receive water that is only moderately soft (i.e., hardness between 60 and 90 mg/l as $CaCO_3$) as well as those whose 45ter is not soft but has a low pH or has other risk factors associated with aggressiveness (e.g., high levels of chlorine, dissolved oxygen, chlorides, sulfates, etc.) also run a somewhat elevated risk of exposure to high lead levels in drinking water. However, the data on these circumstances are too sparce to use in estimating populations at risk of high lead levels.

II.B.2. Lead Contamination in New Housing

As was discussed briefly in section A above, many studies have shown that newly-installed lead solder (or pipes) can leach high amounts of lead in a short amount of time. Indeed, in studies such as Sharrett et al. (1982a), the age of the house (a proxy measure of the age of the plumbing) was the variable most closely related to the lead concentration in the house water. Table II-3 presents some field data on lead levels in new housing. As can be seen, the highest lead contamination levels occur with the newest solder (i.e., during the first 24 months following installation those levels decline and generally are not elevated beyond five years (cf also, Sharrett et al., 1982a; Lassovszky, 1984; etc.).

TABLE II-3 Lead Contamination Levels in Tap Water by Age of Plumbing (Field Studies)

Study	Age of Housing	Mean Pb Level (ug/l)	% of Samples >20 ug/l	Conditions/Notes
Sharrett et al. (1982a)	<18 months	74	NG	(Median standing levels
	<5 years	31	NG	(No lead pipes
	>5 years	4.4	NG	(
Nassau County (1985)	unoccupied	2,690	NG	(Average, first flush
	<2 years	540	NG	(
	2-10 years	60	NG	(
	>10 years	10	NG	(
Philadelphia (1985)	<2 years	90	NG	Flushed
		5000	NG	First-flush
	>2 years	60	NG	Flushed
		500	NG	First-flush
	>4.5 years	<25	NG	NG
EPA (1987) preliminary results	<2 years	NG	93%	First draw (pH <6.4)
		NG	51%	Flushed, 2 minutes (pH <6.4)
	<2 years	NG	83%	First draw (pH 7.0-7.4)
		NG	5%	Flushed, 2 minutes (PH 7.0-7.4)
	<2 years	NG	72%	First draw (pH >8.0)
		NG	0%	Flushed, 2 minutes (pH >8.0)
	2-5 years	NG	84%	First draw (pH <6.4)
		NG	19%	Flushed, 2 minutes (PH <6.4)
	2-5 years	NG	28%	First draw (pH 7.0-7.4)
		NG	7%	Flushed, 2 minutes (pH 7.0-7.4)
	2-5 years	NG	18%	First draw (pH >8.0)
		NG	4%	Flushed, 2 minutes (pH >8.0)
	>5 years	NG	51%	First draw (pH <6.4)
		NG	4%	Flushed, 2 minutes (PH <6.4)
	>5 years	NG	14%	First draw (pH 7.0-7.4)
		NG	0%	Flushed, 2 minutes (PH 7.0-7.4)
	>5 years	NG	13%	First draw (pH >8.0)
		NG	3%	Flushed, 2 minutes (pH >8.0)

KEY: Pb=Lead NG= Not given

Oliphant (1982 and 1983), studying the galvanic action between lead solder and copper pipes, concluded that with any amount of new (exposed) solder alone (i.e., without lead pipes) and sufficient time, lead levels will always eventually exceed 100 ug/l if the volume of the sample is small. The parameters of the water (including pH and total carbonate) are irrelevant to the prediction. His analysis also showed that this galvanic action can produce lead contamination levels 100-1,000 times higher than equilibrium models would predict for the water itself. Over time, however, protective passivation films usually build up in the plumbing and such galvanic couples can stabilize eventually at between 10 and 50 percent of the highest (initial) leaching rates (Oliphant, 1983) or even lower (Lyon and Lenihan, 1977). With copper pipes and new solder, flushed water samples can exceed the current MCL (Philadelphia, 1985; Nassau, 1985; Kuch and Wagner, 1983), although this is not common; the length of new plumbing is probably a significant factor here.

While new housing containing lead solder clearly represents a significant risk of extremely high lead levels in drinking water, the possibility existed that this risk was included in the samples collected by Culligan. We evaluated this in two ways. First, we compared the incidence rate in the Culligan data (16 percent of the samples exceeded 20 ug/l) against the rates presented in studies of new plumbing (about 100 percent > 20 ug/l). The literature on the leaching potential of new plumbing shows much higher contamination levels than is evident in the Culligan kitchen tap samples. Therefore, the data indicate that it is unlikely that these samples included new housing. Second, we

contacted Culligan dealerships in eight different areas, including both areas of recent rapid growth and areas with relatively stable growth patterns. In each office, we asked the representative with the longest sales record to describe the average Culligan customer on a public water supply and to estimate how many customers on public water supplies lived in new homes. The representatives contacted had served many thousands of customers and all described their customers as generally calling Culligan with long-standing problems; for residences connected to community water systems, new problems and problems in new homes were perceived as going first to the local public water utility.

Inhabitants of new housing (i.e., built within the past two years), therefore, represent a separate group at risk of receiving water that exceeds 20 ug/l. Because this population is not represented in the samples of partially flushed kitchen tap water, they must be added to the estimate of exposure.

II.C. Estimated Exposure to Lead in U.S. Tap Water

The estimate of exposure to lead levels in U.S. drinking water > 20 ug/l has several components: 1) the general risk of high lead levels due to the corrosivity of all water and the contact time between tap water and any materials containing lead, 2) the specific risk to inhabitants of new homes (built within the past two years), and 3) many assumptions, including the generalizability of the findings, the distribution of the at-risk populations, the relationship between lead levels in water and human blood-lead levels, compliance with a new regulation, and other issues.

The first part of this section describes some of the many uncertainties and assumptions included in this analysis. The second part presents estimates of potential exposure to tap water containing 20 ug/l of lead or greater.

II.C.1. Uncertainties and Assumptions in the Analysis

It is important to note some of the assumptions and uncertainties that are both inherent and explicit in this analysis.

Assumption: Total compliance. This analysis assumes that, should the MCL be reduced, all community water systems will comply with the new standard by whatever means are necessary for that particular system. In reality, if some systems do not comply, both the costs and the benefits will be overestimated proportionately; the benefit to cost ratio will remain the same because both are functions of the number of people affected.

No more than borderline compliance with the new standard is assumed, however. That is, if the MCL is 20 ug/l, we assume that any systems currently exceeding that level will take measures to reduce their water to that level. If some systems act to reduce lead levels further, both the costs and the benefits will be higher; it is unclear what the ratio between the costs and benefits would be for the incremental reduction.*

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* The case study of Boston (Jacobson, 1986), summarized in Chapter I and appended to this document, indicates that in particular circumstances, the benefit to cost ratio may be quite high -- 11:1 in that case. It is unclear, however, whether or how Jacobson's results can be extrapolated to other U.S. water systems and cities and, therefore, to this proposed rule.

It is unlikely, though, that there is any reasonable and practical scenario in which a water system would institute corrosion control measures where the incremental costs exceed the incremental benefits. At a minimum, therefore, these costs and benefits should be assumed to be equal.

Assumption: Treatment can generally reduce lead levels to 20 ug/l. In general, the data on corrosion control treatment show significant reductions in lead levels (e.g., Karalekas et al., 1977, 1978, 1983; Herrera et al., 1983). However, efforts have generally focused upon reducing lead contamination to levels below the current MCL of 50 ug/l. There is very little data on reducing lead levels below that. Some preliminary data is available from EPA's "Lead Solder Aging Project." These results, shown as Table II-4, indicate that simply raising pH alone greatly reduces the occurrence of samples exceeding 20 ug/l.

Field data from Patterson and O'Brien (1979), Britton and Richards (1981) and others show that adjusting the alkalinity of the water also significantly affects plumbosolvency. Other studies (e.g., Schock and Gardels, 1983) adjusted the level of dissolved inorganic carbonate (DIC) as opposed to alkalinity, because DIC is independent of pH while alkalinity is not. These studies found that increasing DIC could slow lead leaching rates. Some laboratory, experimental, and theoretical analyses have addressed the effect of simultaneously altering several water parameters (e.g., pH, alkalinity, inorganic carbonate, etc.) on plumbosolvency (e.g., Schock, 1980; Jackson and Sheiham, 1980; Schock and Gardels, 1983; Sheiham and Jackson, 1981; Kuch and Wagner, 1983).

However, no treatment has yet been shown to be completely successful in preventing all contamination of drinking water by lead. In particular, new (exposed) plumbing containing lead

TABLE II-4. Percentage of Samples Exceeding 20 ug/l of Lead at Different pH Levels, by Age of House

Age of House	ph	Percent of samples >20 ug/l First-flush	Fully-flushed (2 min)
0-2 years	≤6.4	93%	51%
	7.0 - 7.4	83%	5%
	≥8.0	72%	0%
2-5 years	≤6.4	84%	19%
	7.0 - 7.4	28%	7%
	≥8.0	18%	4%
6+ years	<6.4	51%	4%
	7.0 - 7.4	14%	0%
	<8.0	13%	3%

Source: U.S. EPA (1987), preliminary results from “Lead Solder Aging Study"

seems to continue to produce relatively high levels of contamination even in relatively non-corrosive waters (e.g., Neilsen, 1976). The preliminary results from the EPA solder study on pH adjustment, however, indicate that even these levels can be reduced significantly through treatment.

For this analysis, we assumed that the best treatment currently available will be adequate to reduce lead levels to 20 ug/l, except perhaps in certain specific circumstances such as first-flush waters that have been standing for 16 hours or more, or with lead solder that is under two years old.

Assumption: EPA will change its monitoring requirements to better detect corrosion by-products in drinking water. The common interpretation of EPA's current monitoring requirements calls for fully-flushed samples, typical of distribution water.* Such practice will not capture the presence of lead in drinking water, or indeed, the presence of any corrosion by-products, and results in underestimations of contamination and exposure. This analysis assumes that EPA will change its regulations to capture that exposure and that the criteria for compliance with the new MCL will consider the risk of contamination by corrosion by-products. A revision of EPA's monitoring requirements was called for by the court in the decision on a lawsuit brought by the Environmental Defense Fund against the Agency, as EPA accumulated data on the

* This issue is discussed at the beginning of Section II.B.

factors likely to affect lead levels in drinking water. EDF v. Costle, 578 F. 2d 337, 349-350 (D.C. Cir. 1978). It was also clearly the intent of the National Primary Drinking Water Regulations (40 CFR §141.2(c)) and the Preamble to the Regulations (FR, volume 40, number 248, p. 59575 - December 24, 1975) that corrosion by-products be addressed. These revisions have been expected by the regulated community (e.g., AWWA Committee Report, 1984) and by professionals in the field (e.g., Hoyt et al., 1979; Patterson and O'Brien, 1979) for several years.

Assumption: Adults consume 2 liters of water (or water-based fluids) per day and children consume 1 liter per day. For consistency with past analyses, this document used the estimates that are commonly held in the published literature and that have served as exposure indexes in past EPA actions.

Assumption: Lead levels are tap specific. Because the level of lead contamination depends largely upon the length of contact time between the water and the plumbing as well as the parameters of the water, the particular materials of the private and public plumbing systems, and the age of the plumbing, lead levels vary from tap to tap within a system and even within a house.

Assumption: The relationship between lead in drinking water and human blood-lead levels. The published literature presents several possible equations relating lead levels in human blood to intake of lead from drinking water. This analysis uses the

formulae presented in the Quantification of Toxicological Effects Section of EPA's Water Criteria Document (1985) for lead, which are taken from the Air Quality Criteria Document (U.S. EPA, 1986):

(for children) PbB* = 0.16** x intake of lead from water

(for adults) PbB* = 0.06** x intake of lead from water.

The coefficient for children is taken from Ryu (1983), and the source of the coefficient for adults is Pocock (1983). Both assumed a linear relationship between lead in drinking water and blood lead levels.

Two other general approaches exist. The constant used for children (PbB = 0.16 x Pb in water) is derived from a study of infant blood lead levels (Ryu, 1983) , in which the "constant" was really a non-steady state value. This may be an inappropriate value to use or may be, at best, a lower bound estimate.† A better "constant" may be the steady state value from the control group in the Ryu study, which was 0.45. If so, the projections of children's health effects in this analysis may be underestimated by as much as a factor of 3.

Other studies of the relationship between blood lead levels and water lead concentrations (cf. the discussion and bibliographic citations in the Air Quality Criteria Document, 1986, p. II-106ff.)

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* PbB = blood lead level

** These constants have a unit of ug/dl per ug/day.

† The authors of the Air Quality Criteria Document and the Water Criteria Document are aware of this problem.

for both children and, more commonly, adults, have found cube root functions, typically with intercepts of 4-7 ug/dl (blood lead, with water lead = 0). While the linear forms cited above and used in this analysis provide results similar to the non-linear forms over a very wide range of values (up to, say, 300 ug/l of water), the form of the model greatly influences the estimated contributions to blood lead levels from relatively low water-lead concentrations. Over the typical range of lead levels in U.S. drinking water (0-100 ug/l), the differences in estimated blood lead levels can be quite large. Indeed, in the range of this analysis (generally, water lead levels of 0-50 ug/l) , various cube-root functions yield values that are 4-10 times greater than the estimates using the linear form presented above. Alternative assumptions (e.g., those reasonably derived from the results of Richards and Moore, 1982 or 1984) could indicate that exposure -- and consequently benefits -- in this analysis may be underestimated, possibly by several factors.

Studies investigating the relationship between lead in drinking water and lead in the blood have found a better fit between blood lead levels and lead levels in standing or first-flush samples than other measures of lead contamination of water, for instance, fully flushed samples (Worth et al., 1981; Pocock et al., 1983; Bailey and Russell, 1981). This seems counter-intuitive at first because the intake of standing water would be expected to be much less in total volume consumed than for other water (e.g., partly or fully flushed water), so the expected

contribution of the relatively high lead levels typical of standing samples should be small. But several studies (Rabinowitz et al., 1976 and 1980, in particular) have shown that the absorption of lead varies depending on the state of the gastrointestinal system. Specifically, lead ingested on an empty stomach (e.g., at breakfast or between meals) has a much higher absorption rate than does lead ingested on a full stomach. This could explain the closer correlation between blood lead levels and lead levels in standing or first-flush water.

Where studies have related blood lead levels to several different measures of drinking water lead, the correlation coefficient is larger for the running samples than for the first-flush samples, generally by a factor of 1.5-2 (e.g., Worth et al., 1981; Central Directorate on Environmental Pollution Study, 1982). Whatever measure of water lead is used (first-flush, fully-flushed, etc.), the corresponding coefficient of relationship to blood lead must be used. That means, if lead levels in partly flushed water are measured, the coefficient should also be for partly flushed samples.* However, no studies calculated the relationship between blood lead levels and lead levels in partly flushed water. This analysis, therefore, uses the best available analyses, the Ryu and Pocock studies discussed above, which evaluated first-flush water. The use of the coefficients for first-flush

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* No study has yet calculated a value relating blood lead levels to an integrative measure of water consumption patterns, i.e., a measure reflecting actual drinking habits. A new epidemiological study to derive such a coefficient would be necessary in order to use data on actual consumption patterns.

water with occurrence data relating to contamination levels in partly flushed water introduces a downward bias to the estimates.

In general, the studies assessing the form of the blood-lead/ drinking-water-lead relationship, especially the British studies, assume no contribution to the body burden of lead from any other environmental sources besides drinking water. (The major exception is Worth et al., 1981.) For infants, this may be a less significant omission than for toddlers or adults. Curiously, the authors of these studies do not question why there is an intercept of 4-7 ug/dl, even if water lead is zero. However, gasoline lead is an important determinant of human blood-lead levels (cf. Air Quality Criteria Document for Lead, 1986, p. 11-42ff; Chapter 3 of The Costs and Benefits of Reducing Lead in Gasoline, 1985; and sources cited there) . Indeed, the reduction of gasoline lead levels in the United States in the late 1970s appears to have resulted in a reduction in children's blood-lead levels of almost half during that period (Annest et al., 1983). Lead paint, under certain conditions, can also result in high localized contamination levels.

Analyses of the contributions from various media to human blood-lead levels, focusing upon exposures typical in this country, indicate that drinking water lead may account for about 14-55 percent of the total burden of lead.

It is most likely that drinking water contributes 15-40 percent of the lead body burden (cf, discussions throughout the Air Quality Criteria Document, summarized pp. 13-26ff).

This analysis employs the Ryu and Pocock coefficients (discussed above) relating lead levels in drinking water to blood lead levels; these are combined With the drinking water lead levels presented in this chapter to calculate the potential effect upon blood lead levels. These changes are projected onto extrapolations of the data from the Second National Health and Nutrition Examination Survey (NHANES II)* on the distribution of blood lead levels in the country (cf, Air Quality Criteria Document, chapter 11) to predict the health benefits that would result from a potential reduction in the MCL from 50 ug/l to 20 ug/l.

Assumption: New solder containing lead (under 24 months) contributes an average of 25 ug/l of lead to drinking water. Many field and laboratory studies have found that lead solder alone -- when used with copper household plumbing -- could easily produce lead levels in drinking water well above the current MCL, even in relatively non-corrosive waters. To be conservative, we assumed

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* The NHANES II was a 10,000 person representative sample of the U.S. non-institutionalized population, aged 6 months to 74 years. The survey was conducted by the (U.S.) National Center for Health Statistics (NCHS) over a 4-year period (1976-1980). The data base is available from NCHS and analyses of the lead-related data from it have been published before (e.g., Annest et al., 1982 and 1983; Mahaffey et al., 1982a and 1982b; Pirkle and Annest, 1984).

These extrapolations incorporate the reductions in exposure resulting from the current phasedown in lead in gsoline (the limit is currently 0.1 grams of lead per gallon of gasoline). The projections developed in support of that rule-making and presented in The Costs and Benefits of Reducing Lead in Gasoline (U.S. EPA, 1985b) form the basis of the projections developed for this potential rule.

that new solder would on average produce lead levels at half the MCL, i.e., 25 ug/l, in partly flushed tap water.

Assumption: People drink partly flushed tap water. * Lead levels are highest in first-flush samples, that is, in water that has been sitting for several hours or more (for instance, overnight or all day). But those conditions occur only infrequently (at most, once or twice per day for each faucet), and the sparce data available on actual drinking water use patterns indicate that the bulk of consumed water is partly flushed (e.g., Bailey and Russell, 1981). The likelihood of flushing the water before using it probably follows age and sex patterns, and those most at risk of lead's adverse health effects -- children -- may be least likely to flush the water.

Therefore, EPA has concluded that people are more likely to consume partly flushed water than fully flushed water.

On the other hand, two particular demographic trends over the past few decades are likely to result in an increase in the amount of lead from drinking water to which people are exposed. First, the number and proportion of women working outside the home has increased from 40 percent in 1970 to 51.9 percent in 1983 (Statistical Abstracts, 1985; Tables 27, 658, 659, and elsewhere). This means that more homes will have two 'first-flushes' per day -- one in the morning and the other when the

* Or its equivalent: Some first-flush and some fully flushed water.

parent(s) return from work.* Obviously, this doubles the possibility of exposure to high first-flush lead levels. The second demographic trend that is significant is the decrease in the average number of occupants per housing unit. In 1950, there were, on average, 3.37 people per housing unit, which has decreased to 2.73 in 1983 (Statistical Abstracts, 1985; Table 54). The number of occupants of a dwelling is inversely proportional to lead levels in the drinking water, probably because fewer occupants mean the water will, on average, remain in the pipes longer.

In addition, as noted above in the discussion of the relationship between blood lead levels and levels of lead in drinking water, blood lead levels correspond best with first-flush or standing levels.

Uncertainty: Factors affecting lead levels in multi-family housing, especially high-rise buildings. The available data on lead contamination at the tap comes primarily from single-family homes. It is unclear how lead concentrations in multi-family housing will vary. In the absence of good data, there are three hypothesized possibilities: lead levels will be higher in high-rises than in single-family homes with similar water, lead levels will be lower, or contamination levels and patterns will be the same. There are plausible arguments to support each thesis.

Lead levels may be higher in high-rise buildings because it is more difficult, and perhaps impossible, to fully flush the

* Exposure to lead in the work place or at school is not included in this analysis.

water in a building that is more than a few stories. Because the water is never really flushed, average residence time in pipes is longer and therefore contamination levels will be greater.

On the other hand, lead levels may be lower because one of the factors contributing to high lead levels in tap water is the relatively close ratio of water volume to surface area of pipe resulting from the narrow pipes typical of home plumbing. While the pipes going to each faucet may be comparable, high-rise buildings have service pipes that are, on average, much wider than the largest pipes in homes. Because of the reduced ratio of water to pipe surface area, the potential for high lead contamination may be lower. Another factor, number of occupants, also correlates inversely with lead levels. Because multi-family housing has more occupants in total, average residence time for water in pipes may be shorter than in single family homes.

The third possibility is that both arguments above are correct, and that they cancel out. Contamination levels in large apartment buildings would be comparable to levels in single-family homes.

II.C.2. Calculations of Exposure to Lead in Drinking Water

Several adjustments to the available data on lead levels in tap water and in new housing are necessary to predict the number of people served by community water supplies who are likely to be exposed to drinking water exceeding an MCL of 20 ug/l. These include the assumptions discussed above and other data on the

composition of the housing stock in the United States and the use of various plumbing materials.

These estimates are for one sample year only, 1988. Because the ban on the use of materials containing lead in public water supplies will become enforceable after June 1988, exposure to lead in new housing can be expected to begin to decrease thereafter.

II.C.2.a. Estimate of Exposure to Lead in Drinking Water to Inhabitants of New Housing

The published literature shows that inhabitants of new housing are at risk of exposure to high levels of lead in drinking water. The rates are highest for the first two years, but they decline and are generally not elevated beyond five years.

There were 1.7 million new housing starts and permits in the United States during 1983 and 1.8 million in 1984.* Construction data show that housing typically takes six months to a year from permit to potential occupancy, so there are currently about 3.5 million new housing units (i.e., < 24 months). The Statistical Abstract of the United States (1985) indicates that in 1983, the average household contained 2.73 individuals (Table 58). Multiplied together, a total of 9.6 million people currently live in new housing.

However, not all of these people are served by community water supplies. Of the current (1985) U.S. residential population

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* Survey of Current Business, U.S. Department of Commerce-Bureau of Economic Analysis, 1985; Table on New Housing Construction.

(a little over 240 million), 219.2 million are served by community water systems and this analysis only addresses that population. In addition, the use of plastic plumbing materials has increased recently and voluntary switching to lead-free solder has occurred in many areas; these homes are obviously at decreased risk of exposure to lead from the leaching of new lead/tin solder. Data from the plumbing supply industry show that about 8 percent* of new plumbing is plastic,** so 92 percent of the population has metal pipes. We assumed that virtually all those with metal pipes also have some solder or other fittings containing lead, although this may overestimate exposure somewhat. We assumed that the inhabitants of new housing are distributed proportionately between community and non-community water supplies. Therefore, the number of people served by community water supplies at risk of high lead levels from new solder in new housing is:

$$9.6 \text{ mil} \times \frac{219 \text{ million}}{240 \text{ million}} \times .92 = 8.1 \text{ million people.}$$

This estimate, based upon the current population and current building practices, is for one sample year, 1988. The ban on the future use of materials containing lead in public water supplies

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* This is the arithmetic average of claims by the Plastic Pipe Institute presented in Mruk (1984) and of the Copper Development Association presented in Anderson (1984).

** Plastic pipes are used more commonly for waste water than for intake water. But the use of plastic pipes for both is increasing rapidly.

and in residences connected to them, established by the Safe Drinking Water Act as amended in 1986, will be enforceable after June 1988. Exposure to lead in new housing can be expected to decrease thereafter.

II.C.2.b. Estimate of Exposure to Lead in Drinking Water to Inhabitants of older Housing

The data on partly flushed kitchen tap samples indicate that 16 percent of the drinking water in housing older than two years in this country may exceed an MCL of 20 ug/l. To avoid double counting, the inhabitants of new housing served by community drinking water systems must be subtracted from the total number of people served by community water supplies.

Of the current (1985) U.S. residential population of a little over 240 million, 219.2 million are served by community water systems. Again, we assumed that the people who live in new housing (9.6 million) are distributed proportionately between community and non-community water supplies. Therefore,

$$9.6 \text{ million} \times \frac{219 \text{ million}}{240 \text{ million}} = 8.8 \text{ million people.}$$

To calculate the risk to inhabitants of older housing, subtract the number in new housing (8.8 million) from the total served by community water systems (219.2 million); that indicates that 210.4 million people live in older homes. Based upon the Culligan data, 16 percent of them (33.7 million) are at-risk of high lead levels in partly flushed water at their kitchen taps.

II.C.2.c. Total Estimated Exposure to Lead in Drinking Water

Combining the available data on lead levels in older housing (33.7 million people exposed to lead levels $\geq$ 20 ug/l) with the new housing exposure estimates (8.1 million people at risk) indicates that 41.8 million people using public water supplies currently may be exposed to some water that exceeds the proposed MCL of 20 ug/l; we round this to 42 million.

This estimate is for one sample year, 1988. Many uncertainties surround this estimate, indicating that it may be high or low. Overall, exposure to lead in drinking water is expected to decrease somewhat after 1988 because of the Congressional ban on the future use of pipes, solder and flux containing lead in public water systems and in residences connected to them.

On the other hand, this may be a low estimate

- because it does not include the potential exposure of occupants in housing built within the past 2-5 years;*
- because we have not included those who, while living in older housing, have recently had major plumbing repairs and so are also at risk of the potentially high lead levels associated with newly-installed solder;
- because the Culligan data represent water that is harder than average, whereas high lead levels are often found with soft waters;

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* The incremental risk to inhabitants of 2-5 year old housing is not included in this analysis because it was not possible to eliminate those people from the base and thus avoid double-counting.

- because the data used are for partially flushed samples, while some people (especially children) may consume water that is closer to first-flush or standing samples;* and

- because some of the statistical and analytical techniques used lend a downward bias to the results (e.g., the method of sample preservation and the use of a first-flush correlation coefficient with data on lead levels in partly-flushed water).

In addition, we have not included any data from the estimated 60 million people served wholly or in part by private and non-community water supplies.**

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* Blood lead levels are more closely related to lead levels in first flush or standing water.

** Some people are served by both public and private supplies. For instance, a person's home may be served by a public water system while the drinking water supply at school or work may be private.

CHAPTER III

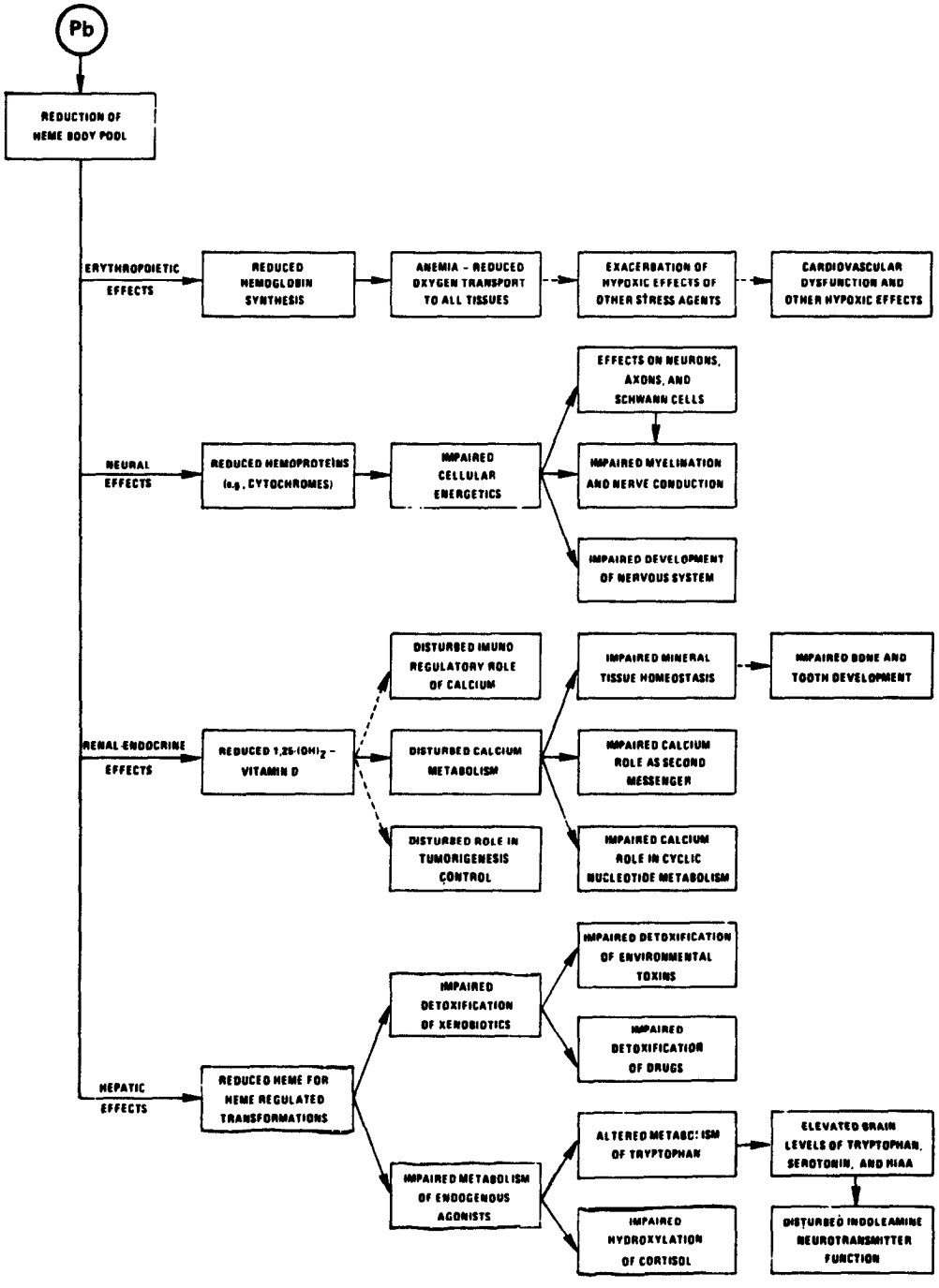
BENEFITS OF REDUCING CHILDREN'S EXPOSURE TO LEAD

The scientific literature presents evidence of a variety of physiological effects associated with exposure to lead, ranging from relatively subtle changes in various biochemical measurements at very low levels of exposure, to severe retardation and even death at very high levels of exposure. Although such effects are found in individuals of all ages, particular concern has focused on children.

Because the body is a complex structure of interdependent systems and processes, effects upon one component will have cascading implications throughout the body. This interdependence is well illustrated by multi-organ impacts resulting from the inhibition of heme synthesis by lead, with consequent reduction in the body heme pool. These effects are depicted graphically in Figure III-1, taken from EPA's most recent Air Quality Criteria Document for Lead (1986; p. 13-31). A summary of children's health effects from exposure to lead, taken from the Air Quality Criteria Document and included in the Water Criteria Document for Lead (1985), p. VIII-65, is also included here as Figure III-2.

This chapter summarizes the available evidence of the effects of lead on children, and estimates some of the health benefits of reducing exposure by reducing lead concentrations in drinking water. Section A deals with the pathophysiological effects of lead, while Section B addresses the evidence on neuropsychological effects (primarily reduced cognitive ability), and Section C discusses the fetal effects of lead exposure. Section D presents

Figure III-1 Multi-Organ Impacts of Lead's Effects on the Heme Pool



Multi-organ impact of reductions of heme body pool by lead. Impairment of heme synthesis by lead results in disruption of a wide variety of important physiological processes in many organs and tissues. Particularly well documented are erythropoietic, neural, renal-endocrine, and hepatic effects indicated above by solid arrows (⟶). Plausible further consequences of heme synthesis interference by lead which remain to be more conclusively established are indicated by dashed arrows (⇢).

FIGURE III-2. SUMMARY OF LOWEST OBSERVED EFFECT LEVELS FOR KEY LEAD-INDUCED HEALTH EFFECTS IN CHILDREN

Lowest Observed Effect Level (PbB)	Heme Synthesis and Hematological Effects	Neurological Effects	Renal System Effects	Gastrointestinal Effects
80-100 ug/dl		Encephalopathic signs and symptoms	Chronic nephropathy (aminoaciduria, etc.)	Colic, other overt gastrointestinal symptoms
70 ug/dl	Frank anemia			
60 ug/dl		Peripheral neuropathies		
50 ug/dl		?		
40 ug/dl	Reduced hemoglobin synthesis	CNS cognitive effects (IQ deficits, etc.)		
	Elevated coproporphyrin	Peripheral nerve dysfunction (slowed NCV's)		
	Increased urinary ALA			
30 ug/dl			Vitamin D metabolism interference	
15 ug/dl	Erythrocyte protoporphyrin elevation	Altered CNS electrophysiological responses		
10 ug/dl	ALA-D inhibition			
	Py-5-N activity inhibition	?	?	
	?			

Abbreviations: PbB = blood lead concentrations; Py-5-N = pyrimidine-5'-nucleotidase; CNS = central nervous system; NCV = nerve conduction velocit; ALA = aminolevulinic acid

Source: Air Quality Criteria Document for Lead (1986)

the methods used to monetize the benefits of reducing children's exposure to lead. Section E discusses some inherent limitations of cost-of-illness studies, upon which most of these monetized health benefits are based, and lists many additional health effects that are not included at all in our analysis. A summary of the monetized and non-monetized children's health benefits for one sample year (1988) is presented in Section F.

Fuller discussions of the adverse health effects associated with lead exposure can be found in other EPA documents: the Air Quality Criteria for Lead (1986), including the Addendum which is part of Volume 1; the Quantification of Toxicological Effects section of the Drinking Water Criteria Document on Lead (1985); and The Costs and Benefits of Reducing Lead in Gasoline (1985).

This document rests heavily upon the analysis and methodologies in The Costs and Benefits of Reducing Lead in Gasoline. Two sections (III.A.4 on Stature Effects and III.C. on Fetal Effects) have been expanded from the earlier analysis; the other sections have been condensed. This reflects the inclusion of new materials and is not an indication of relative importance. In addition, this document includes an alternative method for valuing one aspect of cognitive damage: potential decrement in IQ, valued as a function of the potential decrease in future earnings. This chapter also contains a discussion of the limitations of cost-of-illness studies, both in general and of the specific studies which serve as the basis of this analysis, that did not appear in the earlier cost benefit analysis.

The estimates of health benefits associated with this proposed rule rely upon data on the distribution of lead levels in children and adults collected as part of the Second National Health and Nutrition Evaluation Survey (NHANES II). The NHANES II was a 10,000 person representative sample of the U.S. non-institutionalized population, aged 6 months to 74 years. The survey was conducted by the (U.S.) National Center for Health Statistics (NCHS) over a four-year period (1976-1980). The data base is available from NCHS and analyses of the lead-related data from it have been published before (e.g., Annest et al., 1982 and 1983; Mahaffey et al., 1982a and 1982b; Pirkle and Annest, 1984). This survey provides careful blood, biochemical, nutritional and many other biological, social, and demographic measures representative of the U.S. population.

The fact that other sources of lead, especially gasoline, would slowly decline even without new EPA drinking water standards created a slight complication in projecting blood lead levels for sample year 1988. Because gasoline lead levels fall over time as unleaded gasoline replaces leaded, the difference in blood lead levels resulting from this rule will change over time. The estimates in this report account for both reductions in some other sources of lead and changes in the demographic profile of the U.S. population. This model served EPA also in its analytical efforts supporting the most recent phasedown in the amount of lead permitted in leaded gasoline; it is discussed more fully in The Costs and Benefits of Reducing Lead in Gasoline (US-EPA, 1985b).

III.A. Pathophysiological Effects

Elevated blood-lead levels have long been associated with neurotoxicological effects and many other pathological phenomena: an article on lead's neurotoxicity was published as early as 1839, on kidney damage in 1862, and on impaired reproductive function in 1860. From an historical perspective, lead exposure levels considered acceptable for either occupationally-exposed persons or the general population have been revised downward steadily as more sophisticated biomedical techniques have shown formerly-unrecognized biological effects, and as concern has increased regarding the medical and social significance of such effects. In the most recent downward revision of maximum safe levels (late 1984 - early 1985), the Centers for Disease Control (CDC) lowered its definition of lead toxicity to 25 ug/dl blood lead and 35 ug/dl of erythrocyte protoporphyrin (EP). The present literature shows biological effects as low as 10 ug/dl (for heme biosynthesis) or even 6 ug/dl (for fetal effects and for IQ effects in some populations); indeed, some effects (e.g., elevated ALA levels, hearing decrements, or stature effects) have exhibited no threshold so far.

There is no convincing evidence that lead has any beneficial biological effect in humans (Expert Committee on Trace Metal Essentiality, 1983).

The finding of biological effects at the lowest observed blood-lead levels (4-6 ug/dl) potentially has important implications for public health, because such levels are common in the

U.S. population. As Table III-1 shows, between 1976 and 1980 over three-quarters of children under the age of 18 had blood lead levels in excess of 10 ug/dl, and 15 percent exceeded 20 ug/dl. The rates among blacks and among preschool children were even higher.

Lead's diverse biological effects on humans and animals are seen at the subcellular level of organellar structures and processes, and at the overall level of general functioning that encompasses all of the bodily systems operating in a coordinated, interdependent way. The biological basis of lead toxicity is its ability, as a metallic cation, to bind to bio-molecular substances crucial to normal physiological functions, thereby interfering with these functions. Specific biochemical mechanisms include lead's competition with essential metals for binding sites, inhibition of enzyme activity, and inhibition or alteration of essential ion transport. The effects of lead on certain subcellular organelles, which result in biochemical derangements common to and affecting many tissues and organ systems (e.g., the disruption of heme synthesis processes), are the origin of many of the diverse types of lead-based functional disruptions of organ systems.

Lead is associated with a continuum of pathophysiological effects across a broad range of exposures. In addition to the high level effects mentioned above, there is evidence that low blood-lead levels result in:

1. Inhibition of pyrimidine-5'-nucleotidase (Py-5-N) and delta-aminolevulinic acid dehydrase (ALA-D) activity, which appears to begin at 10 ug/dl of

TABLE III-1. Blood Lead Levels of Children in the United States 1976-80 (percent in each cell; rows sum to 100 percent)

	<10 ug/dl	10-19 ug/dl	20-29 ug/dl	30-39 ug/dl	40-69 ug/dl
All Races					
all ages	22.1	62.9	13.0	1.6	0.3
6 months-5 years	12.2	63.3	20.5	3.5	0.4
6-17 years	27.6	64.8	7.1	0.5	0.0
White					
all ages	23.3	62.8	12.2	1.5	0.3
6 months-5 years	14.5	67.5	16.1	1.8	0.2
6-17 years	30.4	63.4	5.8	0.4	0.0
Black					
all ages	4.0	59.6	31.0	4.1	1.3
6 months-5 years	2.7	48.8	35.1	11.1	2.4
6-17 years	8.0	69.9	21.1	1.0	0.0

blood lead or below (Angle et al., 1982). Hernberg and Nikkanen (1970) found 50 percent of ALA-D inhibited at about 16 ug/dl.

Inhibition of erythrocyte ALA-D appears to occur at virtually all blood lead levels measured so far, and any threshold remains to be determined (cf, summary of literature in Air Quality Criteria Document, 1986; pp. 12-13 to 12-51).

2. Elevated levels of EP zinc protoporphyrin (ZPP) in red blood cells at about 15 ug/dl. This probably indicates a general interference in heme synthesis throughout the body, including interference in the functioning of mitochondria (Piomelli et al., 1977). Changes in heme metabolism have been reported perinatally at blood lead levels of 8-10 ug/dl (Lauwerys et al., 1978). Some studies that accounted for iron status show that children with low iron stores are more sensitive to lead in terms of heme biosynthesis interference (e.g. , Mahaffey and Annest, 1986).

3. Changes in the electrophysiological functioning of the nervous system. This includes changes in slow-wave electroencephalogram (EEG) patterns and increased latencies in brainstem auditory evoked potentials (Otto et al., 1981, 1982, 1984) which begin to occur at about 15 ug/dl The changes in slow-wave EEG patterns appear to persist over a two-year period. Also , the relative amplitude of synchronized EEG between left and right lobe shows effects starting at about 15 ug/dl (Benignus et al., 1981). Finally, there is a significant negative correlation between blood lead and nerve conduction velocity in children whose blood lead levels range from 15 ug/dl to about 90 ug/dl (Landrigan et al., 1976).

4. Inhibition of globin synthesis, which begins to appear at approximately 20 ug/dl (White and Harvey, 1972; Dresner et al., 1982).

5. Increased levels of aminolevulinic acid (ALA) in blood and soft tissue, which appear to occur at about 15 ug/dl and may occur at lower levels (Meredith et al., 1978). Several studies indicated that increases of ALA in the brain interfered with the gamma-aminobutyric acid (GABA) neurotransmitter system in several ways (Criteria Document, 1986; p. 12-145 ff).

6. Inhibition of vitamin D pathways, which has been detected at the lowest observed blood-lead levels (Rosen et al., 1980a, 1980b; Mahaffey et al., 1982). Further, as blood lead levels increaser the inhibition becomes increasingly severe.

7. An inverse relation between maternal and fetal blood-lead levels and gestational age, birth weight, and early post-natal development (both physical and mental) down to 10 ug/dl and possibly below (Bellinger et al., 1984; McMichael et al., 1986). Investigations of post-natal growth and development also present evidence of a negative association with blood-lead levels at the lowest observed blood-lead level (Schwartz et al., 1986).

8. Finally, recent studies of IQ effects in poor black children (Schroeder, 1985; Schroeder and Hawk, 1986) show IQ effects over the range of 6 to 47 ug/dl without an evident threshold (cf also Air Quality Criteria Document, 1986; p. 12-92 ff, 12-157, and elsewhere). Another recent article (Schwartz and Otto, 1987) shows hearing effects throughout the range of measured blood-lead levels.

These data cite the lowest observed effect levels to date, and do not necessarily represent affirmative findings of thresholds below which exposures can be considered safe.

The specific effects listed above as occurring at blood lead levels below 25 ug/dl indicate (a) a generalized lead impact on erythrocytic pyrimidine metabolism, (b) a generalized lead-induced inhibition of heme synthesis, (c) lead-induced interference with vitamin D metabolism, and (d) lead-induced perturbations in central and peripheral nervous system functioning.

As lead exposure increases, the effects become more pronounced and broaden to additional biochemical and physiological mechanisms in various tissues, causing more severe disruptions of the normal functioning of many organ systems. At very high lead exposures, the neurotoxicity and other pathophysiological changes can result in death or, in some cases of non-fatal lead poisoning, long-lasting sequelae such as mental retardation and severe kidney disease.

This chapter discusses the known pathophysiological effects of lead that occur in children, particularly the neurotoxic and fetal effects, and the expected change in the number of children at potential risk of those effects under EPA's proposed drinking water regulation.

III.A.1. Effects of Lead on Pyrimidine Metabolism

The best-known effect of lead on erythrocytic pyrimidine metabolism is the pronounced inhibition of Py-5-N activity, an enzyme that controls the degradation and removal of nucleic acid from the maturing red blood cell (reticulocyte). As noted earlier, the disruption of this function by lead has been noted at exposure levels beginning at 10 ug/dl. At blood lead levels of 30-40 ug/dl, this disturbance is sufficient to materially contribute to red blood cell destruction and, possibly, decreased hemoglobin production contributing to anemia (World Health Organization, 1977; National Academy of Sciences, 1972; Lin-Fu, 1973; Betts et al., 1973). The mechanism of this inhibition may have a wide-spread impact on all organs and tissues.

III.A.2. Effects on Heme Synthesis and Related Hematological Processes

These effects, are described more fully in the Air Quality Criteria for Lead (EPA, 1986), are only summarized here.

III.A.2.a. Mitochondrial Effects

The mitochondrion is an organelle within the cell and outside the nucleus that produces energy for the cell through cellular respiration and is rich in fats, proteins and enzymes. It is the critical target organelle for lead toxicity in a variety of cell and tissue types, followed probably by cellular and intracellular membranes. The scientific literature shows evidence of both structural injury to the mitochondrion (e.g., Goyer and Rhyne, 1973; Fowler, 1978; Fowler et al., 1980; Bull, 1980; Pounds et al., 1982a and 1982b) and impairment of basic cellular energetic and other mitochondrial functions (e.g., Bull et a1., 1975; Bull, 1977, 1980; Holtzman et al., 1981; Silbergeld et al., 1980). These and other studies also provide evidence of uptake of lead by mitochondria in vivo and in vitro.

III.A.2.b. Heme Synthesis Effects

The best-documented effects of lead are upon heme biosynthesis. Heme, in addition to being a constituent of hemoglobin, is an obligatory constituent for diverse hemoproteins in all tissues, both neural and non-neural. Hemoproteins have important roles in generalized functions, such as cellular energetic, as well as in more specific functions such as oxygen transport and detoxification of toxic foreign substances (e.g., the mixed-function oxidase system in the liver). Statistically significant effects are detectable at 10-15 ug\dl.

The interference of lead with heme synthesis in liver mitochondria appears to result in the reduced capacity of the liver to break down tryptophan, which, in turn, appears to increase

levels of tryptophan and serotonin in the brain (Litman and Correia, 1983). This may account for some of the neurotoxic effects of lead.

The elevation of aminolevulinic acid (ALA) levels is another indication of lead's interference in heme synthesis and mitochondrial functioning. Because increased ALA is associated with significant inhibition of certain kinds of neurotransmission, such elevations can have serious neurotoxic implications. Thus, in addition to its direct molecular neurotoxicity, lead may adversely affect the brain at low exposure levels by altering heme synthesis (e.g., Silbergeld et al., 1982). There appears to be no threshold concentration for ALA at the neuronal synapse below which presynaptic inhibition of GABA release ceases.

Since ALA passes the blood brain barrier and is taken up by brain tissue, it is likely that elevated ALA levels in the blood correspond to elevated ALA levels in the brain (Moore and Meredith, 1976). Furthermore, lead in the brain is likely to enhance brain ALA concentrations because neurons are rich in mitochondria, the subcellular site of ALA production. As mentioned earlier, blood ALA elevations are detectable at 18 ug/dl of blood lead (Meredith et al., 1978).

III.A.3. Lead's Interference with Vitamin D Metabolism and Associated Physiological Processes

Another potentially serious consequence of lead exposure is the impairment of the biosynthesis of the active vitamin D metabolize, 1,25-$(OH)_2$ vitamin D, detectable at blood lead levels of 10-15 ug/dl. Further, an inverse dose-response relationship has been reported between blood lead and 1,25-$(OH)_2$

vitamin D throughout the range of measured blood lead values up to 120 ug/dl (Criteria Document, p. 12-37 ff.; Rosen et al., 1980a, 1980b; Mahaffey et al., 1982b). Interference with vitamin D production disrupts calcium and phosphorous homeostasis, partially resulting in the reduced absorption of these elements from the gastro-intestinal tract. This may alter the availability of these elements for physiological processes crucial to the normal functioning of many tissues, cell membranes, and organ systems.

The reduced uptake and utilization of calcium has two compounding consequences. First, it interferes with calcium-dependent processes that are essential to the functioning of nerve cells, endocrine cells, muscle cells (including those in the heart and other components of the cardiovascular system), bone cells, and most other types of cells. The second concern is possible increased lead absorption resulting from decreased calcium availability. The latter can be expected to further exacerbate the inhibition of vitamin D metabolism and reduced calcium availability (Sorrell et al., 1977; Mahaffey et al., 1986), resulting in even greater lead absorption and greater vulnerability to increasingly more severe lead-induced health effects (Rosen et al., 1980b; Barton et al., 1978). These effects are especially dangerous for young (preschool age) children who are developing rapidly. These children, even in the absence of lead, require a relatively high intake of calcium to support the formation of the skeletal system, as well as several other calcium-dependent physiological processes important in young children.

Even moderate levels of lead exposure in children are associated with vitamin D disturbances that parallel certain metabolic disorders and other disease states, as well as severe kidney dysfunction (Criteria Document, 1986; p. 12-37). At blood lead levels of 33-55 ug/dl, 1,25-$(OH)_2$ vitamin D is reduced to levels comparable to those observed in children who have severe renal insufficiency with the loss of about two-thirds of their normal kidney function (Rosen et al., 1980a; Rosen and Chesney, 1983; Chesney et al., 1983). Analogous vitamin D hormone depressions are found in vitamin D-dependent rickets (type I), oxalosis, hormone-deficient hypoparathyroidism, and aluminum intoxication in children undergoing total parenteral nutrition.

Lead-induced interference with 1,25-$(OH)_2$ vitamin D biosynthesis affects a wide range of physiological processes. The vitamin D-endocrine system is responsible in large part for the maintenance of extra- and intra-cellular calcium homeostasis (Rasmussen and Waisman, 1983; Wong, 1983; Shlossman et al., 1982; Rosen and Chesney, 1983). Thus, modulation in cellular calcium metabolism induced by lead at relatively low concentrations may potentially disturb multiple functions of different tissues that depend upon calcium as a second messenger (Criteria Document, p. 12-40). It also appears that 1,25-$(OH)_2$ vitamin D participates directly in bone turnover by orchestrating the population of cells within the bone (Criteria Document, p. 12-41). An immunoregulatory role for the vitamin D hormone is evident through the widespread existence of 1,25-$(OH)_2$ vitamin D_3 receptor sites in

immunoregulatory cells, such as monocytes and activated lymphocytes (Provvedini et al., 1983; Bhalla et al., 1983).

The negative correlation between blood lead and serum 1,25-$(OH)_2$ vitamin D, the hormonally active form of vitamin D, appears to be another example of lead's disruption of mitochondrial activity at low concentrations. While serum levels of 1,25-$(OH)_2$ vitamin D decreased continuously as blood lead levels increased from the lowest measured level (12 ug/dl), this was not true for its precursor, 25-(OH) vitamin D. In fact, in lead-intoxicated children after chelation therapy, 1,25-$(OH)_2$ vitamin D levels were restored, but the precursor levels remained unchanged (Rosen et al., 1980a, 1980b; Mahaffey et al., 1982). This indicates that lead inhibits renal 1-hydroxylase, the kidney enzyme that converts the precursor to the active form of vitamin D. These observations in children are supported by lead effects on vitamin D metabolism in vivo and in vitro (Smith et al., 1981; Edelstein et al., 1984). Renal 1-hydroxylase is a mitochondrial enzyme system, which is mediated by the hemoprotein, cytochrome P-450. This suggests that the damage to the mitochondrial systems detected at 15 ug/dl and below has uncompensated consequences.

If cytochrome P-450 is being inhibited at the low levels of blood lead that the reduced renal 1-hydroxylase activity suggests, it is possible that other physiological functions related to cytochrome P-450 are also disrupted. For example, reduced P-450 content has been correlated with impaired activity of the liver detoxifying enzymes, aniline hydroxylase and aminopyrine demethylase, which help to detoxify various medications and xenobiotics

and modulate the metabolism of steroid hormones (Goldberg et al., 1978; Saenger et al., 1984).

While cytochrome P-450 inhibition has been found in animals, and in humans at higher lead levels, this has not yet been examined in children at blood lead levels < 25 ug/dl. But the disruption of vitamin D biosynthetic pathways at these levels is suggestive of an effect.

The reduction in heme caused by lead exposure probably underlies the effects seen in vitamin D metabolism. This would explain the similarity in the effect of lead on both erythrocyte protoporphyrin accumulation and decreases in levels of serum $1,25\text{-}(OH)_2D$. It would also indicate a cascade of biological effects among many organ and physiological systems of the body (depicted graphically in Figure III-1). Together, the inter-relationships of calcium and lead metabolism, lead's effects on $1,25\text{-}(OH)_2D$, and the apparent disruption of the cytochrome P-450 enzyme system provide a single molecular and mechanistic basis for Aub et al.'s observation in 1926 that "lead follows the calcium stream."

III.A.4. Stature Effects

Small stature has been identified with lead poisoning for many years (e.g. , Nye, 1929), and is a plausible outcome, given the known biotoxic interaction of lead with calcium messengers, heme-dependent enzymes, and neuroendocrine function. Several new studies provide evidence of a much stronger association

between exposure to lead and subsequent growth and development than was previously thought. These studies of stature effects were not included in The Costs and Benefits of Reducing Lead in Gasoline. The Addendum to the Criteria Document (1986), appended to Volume 1, contains a discussion of the deleterious effect of lead upon various aspects of development and growth, even at the relatively low exposure levels encountered by the general population, i.e., 15 ug/dl and below (p. A-31 to A-56).

III.A.4.a. Effects of Lead on Fetal Growth

Many studies have investigated the effect of intrauterine lead exposure on gestational age, fetal growth and fetal physical development.* The Air Quality Criteria Document (1986; pp. 12-192 to 12-220) and the Addendum to the Criteria Document (1986, pp. A-31 to A-56) contain a full review of these studies.

Several studies examined the relationship between maternal or fetal blood-lead levels and gestational age. Moore et al. (1982), for instance, conducted a cross-sectional study of 236 mother-infant pairs in Glascow, Scotland. Blood lead levels showed a significant negative relation to gestational age, for both maternal and cord lead measures. The blood lead levels were within the normal range and higher, with geometric mean blood-lead

* Many of these studies also show a relationship between blood lead levels (both maternal and fetal) and negative pregnancy outcomes, including early membrane rupture, miscarriages and spontaneous abortions, potential minor congenital anomalies in live births, etc. These and other adverse effects upon the fetus are discussed in Section III.C. of this report.

levels of 14 ug/dl for the mothers and 12 ug/dl for the infants. As an indication of the findings, the mean blood-lead levels for the 11 cases of premature birth (gestational age under 38 weeks) were among the highest and averaged about 21 ug/dl for mothers and 17 ug/dl for infants. In this study, first-flush household water lead levels were positively associated with both maternal and fetal blood-lead levels.

In another recent study of gestational age (McMichael et al., 1986), following 774 pregnancies to completion (live birth, spontaneous abortion, or still birth), women with blood lead levels > 14 ug/dl were over 4 times more likely to deliver pre-term than women with blood lead levels of $\leq$ 8 ug/dl. Excluding cases of still births, the relative risk increased to almost 9.

Other studies have looked at the relationship between prenatal exposure to lead and birth weight or size. Nordstrom et al. (1979b), examining records of female employees of a Swedish smelter, found decreased birth weights related to: 1) employment of the mother at the smelter during pregnancy, 2) distance the mother lived from the smelter, and 3) proximity of the mother's job to the actual smelting process. In a related study (Nordstrom et al., 1978a), similar results were found for infants born to mothers merely living near the smelter.

A more recent paper by Bryce-Smith (1986) found both birth weight and head circumference related to placental lead levels in a cohort of 100 normal infants. Another study (Bellinger et al., 1984), studying mental development in middle class children

up to 2 years old, found a more subtle exposure-related trend in the percentage of small-for-gestational-age infants. Dietrich et al. (1986), presenting interim results, also found that prenatal lead levels were associated with both reduced gestational age and reduced birth weight, which in turn were both significantly associated with reduced neurobehavioral performance at three months.

Some other studies (e.g., Clark, 1977; McMichael et al., 1986*) did not find birth weight statistically significantly related to blood lead levels, however. Nonetheless, “the evidence as a whole from these studies indicates that gestational age appears to be reduced as prenatal lead exposure increases, even at blood lead levels below 15 ug/dl" (Addendum to the Criteria Document, 1986; p. A-45).

Other recent studies of lead’s adverse effect upon physical development have assessed neurobehavioral aspects of child development. These studies are described and evaluated in Section B on lead’s neurological effects. Lead’s adverse impact on gestational age, however, has cascading effects upon subsequent mental development in infants.

III.A.4.b. Effects of Lead on Post-Natal Growth

The first article on lead’s effect on stature (Nye, 1929) observed the incidence of "runting", eye squint and drop foot as physical characteristics of overtly lead-poisoned children.

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* The Addendum to the Criteria Document (1986; p. A-43f) suggests that the findings of McMichael et al. are "not entirely clear with regard to birth weight. The proportion of pregnancies resulting in low-birthweight singleton infants [in the high blood-lead group] . . . was more than twice that for the [low blood-lead group].”

Since then, however, surprisingly few studies have investigated this effect, until quite recently.

In the 1970s, three studies (Mooty et al., 1975; Johnson and Tenuta, 1979; Routh et al., 1979) investigated possible stunting of physical growth as an end point of lead exposure. In the first study, the children in the high-lead group (blood leads of 50-80 ug/dl) were shorter and weighed less than those in the low-lead group (blood leads, 10-25 ug/dl). But the high-lead group was also slightly younger (average age 33 months vs. average age 34 months in the low-lead group) and not racially matched, so it is difficult to determine clearly the relative contribution of lead to the difference in stature. Johnson and Tenuta studied the growth and diets of 43 low-income children and also found a relative decrease in height with an increase in blood lead level. But they did not report the specific racial composition and mean ages of the subjects, nor did they assess the relative contribution of differences in calcium intake or the incidence of pica or other factors. Routh and co-workers found the incidence of microencephaly (defined as head circumference at or below the third percentile for the child's age on standard growth charts) was markedly greater among children with blood lead levels $\geq$ 30 ug/dl than in those with blood lead levels below 29 ug/dl. Again, however, it is not possible to distinguish the relative contribution of lead from other (racial, dietary, etc.) factors that may have affected these children's growth. Despite their individual weaknesses, the three studies together are suggestive of an effect.

Much stronger evidence for the retardation of growth and decreased stature associated with exposure to lead has emerged more recently from animal toxicology studies and the evaluation of large epidemiologic data sets.

About 65 papers on animal experimental studies have been published in the last 10 years that investigated the retardation of growth following low-level exposure during intrauterine life, early post-natal life or both. These studies found decreased body weight at blood lead levels of 18-48 ug/dl with no change in food consumption (e.g., Grant et al., 1980). Deficits in the rate of neurobehavioral development and indications of specific organic or functional alterations were observed at blood lead levels as low as 20 ug/dl (e.g., Fowler et al., 1980). As summarized in the Addendum to the Criteria Document (1986; p. A-51), "it seems very clear [from these animal studies] that low-level chronic lead during pre- and early post-natal development does indeed result in retarded growth even in the absence of overt signs of lead poisoning."

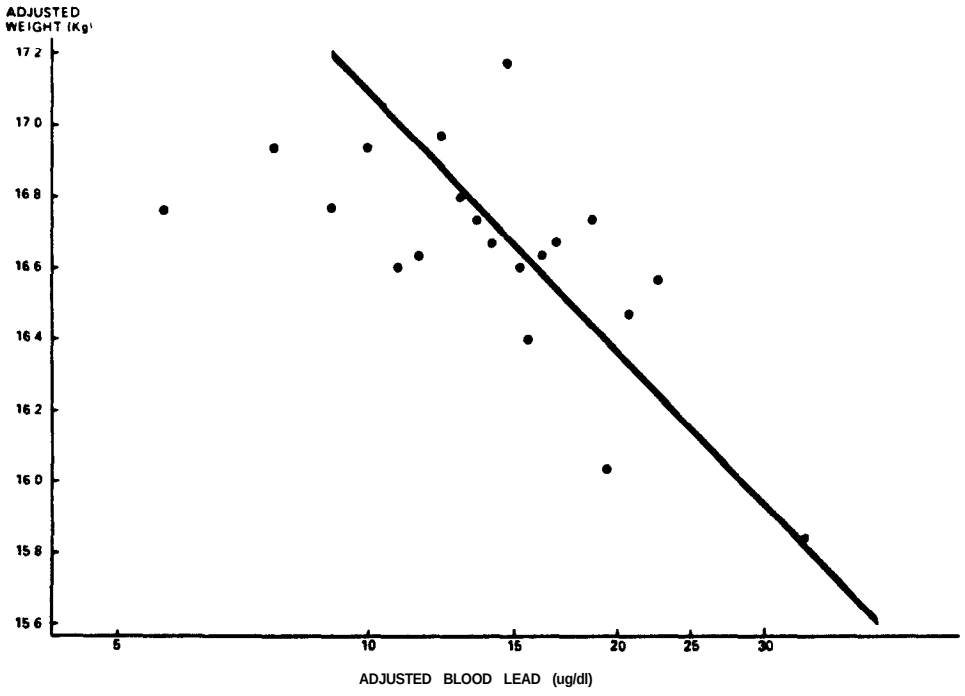
Finally, Schwartz et al. (1986) analyzed results from the Second National Health Assessment and Nutritional Evaluation Survey (NHANES II) to investigate the relationship between blood lead levels and physical development, controlling for other contributing factors, including age, race, sex, several measures of nutritional status, family income, degree of urbanization and many other variables enabling them to account for general health, and environmental and nutritional factors that might not be adequately controlled for by the nutrient and blood measurements.

To assure that blood lead was not found to be significantly associated with growth and development because of the correlation between blood lead and nutritional status, a stepwise regression procedure employing potential confounding variables was used. To address the NHANES II survey design, the computer program SURREGR was used.

Schwartz's results show that blood lead levels are a statistically significant predictor of children's height ($p < 0.0001$), weight ($p < 0.001$) and chest circumference ($p < 0.026$), after controlling for age (in months), race, sex and nutritional covariates.

Figures III-3 and III-4 illustrate the relationship of stature (height and weight) to blood lead, after controlling for all of the other covariates. The threshold regressions (using segmented regression models) indicate that there is no identified threshold for the relationship down to the lowest observed blood lead of 4 ug/dl. The relationship is consistent through the normal range (5-35 ug/dl).

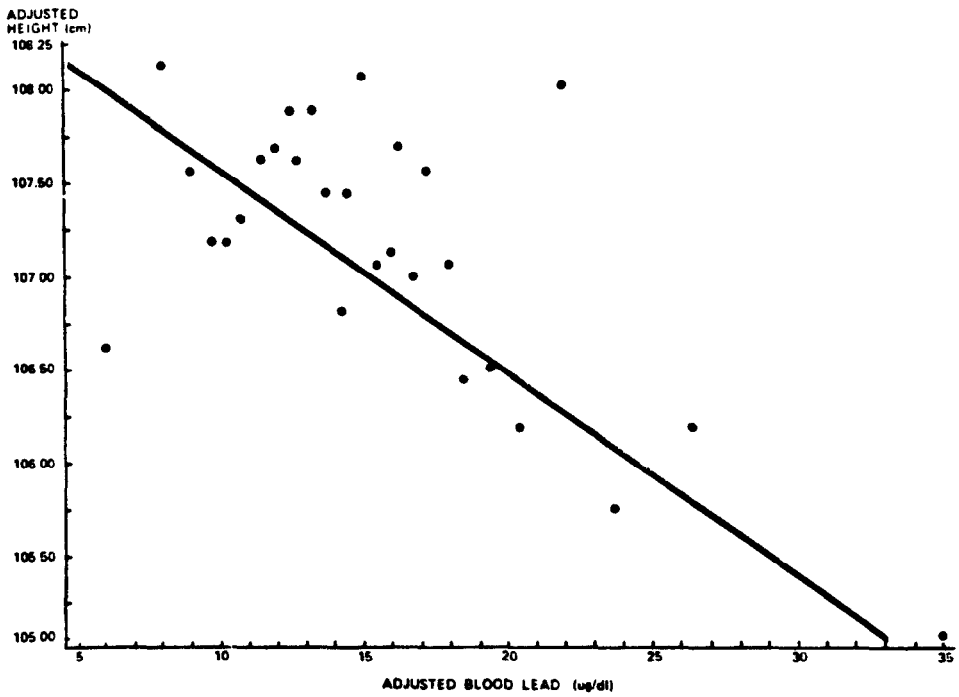
FIGURE III-3 Relationship of Blood Lead Level to Weight in Children Aged 0 to 7



Adjusted weight and adjusted blood lead levels for children aged 7 years and younger in Second National Health and Nutrition Examination Survey. Both weight and blood lead level have been adjusted by regression for effects of age, race, sex, and all other variables significant at .05 level. Each point is mean weight and mean blood lead level of approximately 70 consecutive observations, ordered by blood lead levels. Regression line reflects slope of coefficient obtained from multiple regression analysis of all 1,967 observations with no missing data.

Source: Schwartz et al., 1986.

FIGURE III-4 Relationship of Blood Lead Level to Height in Children Aged 0 to 7



Adjusted height and adjusted blood lead levels for children aged 7 years and younger in Second National Health and Nutrition Examination Survey. Both height and blood lead level have been adjusted by regression for effects of age, race, sex, and all other variables significant at .05 level. Each point is mean height and mean blood lead level of approximately 100 consecutive observations, ordered by blood lead levels. Regression line reflects slope of coefficient obtained from multiple regression analysis of all 2,695 observations.

Source: Schwartz et al ., 1986.

At the average age (59 months), the mean blood-lead level of the children appears to be associated with a reduction of about 1.5 percent in the height that would be expected if their blood lead level was zero. The relative impact on weight and chest circumference is of the same magnitude.

111.A.4.C. Summary of Stature Effects

The inverse correlation of blood lead and growth in U.S. children is often understood in the context that blood lead is a composite factor for genetic, ethnic, nutritional, environmental, and socio-cultural factors that are insufficiently delineated by age, race, sex and nutrition or by family income, urban residence, and all other available nutritional indices. An environment that favors a higher blood lead* may supercede all of the established predictors such as socioeconomic status and other demographic characteristics.

Growth is a complicated phenomenon, accompanied by an orderly sequence of maturational changes. There are many mechanisms that may account for lead's effect on physical growth and development. Prenatal exposure has an inverse effect on gestational age, which

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* Assessments of the risk of ambient lead exposure recognize the triple jeopardy of the urban poor: 1) the exposure to lead from multiple sources is highest in low income areas; 2) in high lead environments, the amount ingested increases with deficiencies in child care and household cleanliness; and 3) the intestinal absorption of lead increases with nutritional deficits. The interaction of socio-cultural and nutritional deprivation with both environmental exposure and absorption of lead has long confounded the delineation of the threshold for behavioral and cognitive effects of low-level lead exposure.

in turn can adversely affect growth. There are known negative interactions between lead and calcium messengers, heme-dependent enzymes, and neuroendocrine function. While the effect is clearly plausible, little research has investigated potential mechanisms directly. At least one very recent article (Huseman et al., 1987) uses a rat pituitary model to support the biological plausibility of a neuroendocrine effect on growth.

At 20 ug/dl, vitamin D metabolism is potentially sufficiently disrupted to hamper the uptake and utilization of calcium, and children are one-and-a-half times more likely to exhibit abnormal red blood cell indices than at 10 ug/dl. For this analysis, we have assumed that children with blood lead levels over 20 ug/dl are at risk of suffering from smaller stature. To assess the benefit of this potential rule, we used the NHANES data on the distribution of blood lead levels in the country to calculate the number of children who would be brought below 20 ug/dl of blood lead at an MCL of 20 ug/l: 82,000.

We have ascribed no monetary value to this health effect because it is difficult to put a monetary value on gestational age, fetal development, and children's growth and stature. The correlation with blood lead level is independent of the significant effects on growth of sex, race, and nutritional status, as well as all identifiable measures of socioeconomic status. As yet, it has exhibited no threshold. Common sense, however, suggests the value would be high.

The method used to calculate the number of fetuses at risk of exposure to potentially dangerous levels of lead is described in Section C, below.

III. B. Neurotoxic Effects of Lead Exposure

Lead has been known to be a neurotoxicant since the early 1800s, and neurotoxicity is among the more severe consequences of lead exposure. At very high blood-lead levels, encephalopathy and severe neurotoxic effects are well documented; the neurotoxic effects at lower blood-lead levels, however, are less clearly defined. Recent research has investigated the occurrence of overt signs and symptoms of neurotoxicity and the manifestation of more subtle indications of altered neurological functions in individuals who do not show obvious signs of lead poisoning.

This section presents new data on cognitive effects at low levels of lead exposure. These studies were not discussed in The Costs and Benefits of Reducing Lead in Gasoline.

III.B.1. Neurotoxicity at Elevated Blood-Lead Levels

Very high blood-lead levels (i.e., above 80 ug/dl in children) are associated with massive neurotoxic effects that can include severe, irreversible brain damage; ataxia (i.e., the inability to coordinate voluntary muscular movements); persistent vomiting; lethargy; stupor; convulsions; coma; and sometimes death. Once encephalopathy occurs, the risk of death for children is significant (Ennis and Harrison, 1950; Agerty, 1952; Lewis et al., 1955), regardless of the quality of the medical treatment they receive.

In cases of severe or prolonged nonfatal episodes of lead encephalopathy, the neurological damage is qualitatively similar to that often seen following traumatic or infectious cerebral injury, with permanent and irreversible damage being more common in children than adults (Mellins and Jenkins, 1955; Chisolm, 1956, 1968). The most severe effects are cortical atrophy, hydrocephalus (an abnormal increase in cranial fluid), convulsive seizures, and severe mental retardation. Permanent central nervous system damage almost always occurs in children who survive acute lead encephalopathy and are re-exposed to lead (Chisolm and Harrison, 1956). Even if their blood lead levels are kept fairly low, 25-50 percent show severe permanent sequelae including seizures, nervous disorders, blindness, and hemiparesis (paralysis of half of the body) (Chisolm and Barltrop, 1979).

Even children without obvious signs of acute lead encephalopathy have exhibited persisting neurological damage. As early as 1943, Byers and Lord’s study of 20 previously lead-poisoned children indicated that 19 later performed unsatisfactorily in school, "presumably due to sensorimotor deficits, short attention span, and behavioral disorders". Effects such as mental retardation, seizures, cerebral palsy, optic atrophy, sensorimotor deficits, visual-perceptual problems, and behavior disorders have been documented extensively in children following overt lead intoxication or even just known high exposures to lead (e.g., Chisolm and Harrison, 1956; Cohen and Ahrens, 1959; Perlstein and Attala, 1966).

The extent of the later manifestations seems to relate to the severity of the earlier observed symptoms. In Perlstein and Attala, 9 percent of the children studied, none of whom appeared to have severe symptoms when diagnosed for overt lead poisoning, were later observed to be minimally mentally retarded and 37 percent showed some lasting neurological sequelae.

At somewhat lower blood-lead levels (i.e., 30-70 ug/dl), substantial data confirm that a variety of neural dysfunctions occur in apparently asymptomatic children. Several studies indicate that blood lead levels of 50-70 ug/dl are associated with IQ decrements of 5 points. Adverse electrophysiological effects, including markedly abnormal EEG patterns, slow-wave voltages, etc., are also well documented at levels of 30-70 ug/dl and even below.

De la Burde and Choate (1972, 1975) showed persisting neuro-behavioral deficits in children exposed to moderate-to-high levels of lead; most of the children appear to have had blood lead levels $\geq$ 50 ug/dl. Compared to low-lead control children -- matched for age, sex, race, parents' socioeconomic status, housing density, mother's IQ, number of children in the family below age 6, presence of father in the home, and mother working -- the higher lead children averaged about five points lower in IQ and were seven times more likely to have repeated grades in school or to have been referred to school psychologists. Moreover, follow-up studies showed that these effects persisted for at least three years.

The 5-point IQ decrement found in asymptomatic children with blood lead levels $\geq$ 50 ug/dl is consistent with other studies.

These include Rummo (1974) and Rummo et al. (1979), which found a 16-point decrement in children > 80 ug/dl and a 5-point loss in asymptomatic children averaging about 68 ug/dl, and reanalysis by Ernhart of the data in Perino and Ernhart (1974) and Ernhart et al. (1981), as described in the Criteria Document (US-EPA, 1986; p. 12-81 to 12-85).*

While chelation therapy may mitigate some of these persisting effects, significant permanent neurological and cognitive damage results from very high lead levels, with or without encephalopathy. In addition, these children also appear more likely to experience neurological and behavioral impairments later in childhood.

III.B.2. Neurotoxicity at Lower Blood-Lead Levels

The adverse effects of lead on neurological functioning, both on the microscopic (e.g., cellular and enzymatic) level and the macroscopic (e.g., learning behavior) level, are well documented. On the micro-level, data from experimental animal studies suggest several possible mechanisms for the induction of neural effects, including: (1) increased accumulation of ALA in the brain as a consequence of lead-induced impaired heme synthesis, (2) altered ionic balances and movement of ions across axonal membranes and at nerve terminals during the initiation or conduction of nerve impulses due to lead-induced effects on the metabolism or synaptic utilization of calcium, and (3) lead-induced effects on the metabolism or synaptic utilization of various neurotransmitters.

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* Ernhart submitted the re-analysis, with better control for confounding variables and with errors corrected to EPA's Expert Committee on Pediatric Neurobehavioral Evaluations (1983).

In addition, lead-induced heme synthesis impairment, resulting in reduced cytochrome C levels in brain cells during crucial developmental periods, has been clearly associated with the delayed development of certain neuronal components and systems in the brains of experimental animals (Holtzman and Shen Hsu, 1976). (Cytochrome C is a link in the mitochondrial electron transport chain that produces energy, in the form of adenosine triphosphate (ATP), for the entire cell.) Given the high energy demands of neurons, selective damage to the nervous system seems plausible.

In addition to the effects of lead on the brain and central nervous system, there is evidence that peripheral nerves are affected as well. Silbergeld and Adler (1978) have noted lead-induced blockage of neurotransmitter (acetylcholine) release in peripheral nerves in rats, a possible result of lead's disruption of the transport of calcium across cellular membranes. This disruption of cellular calcium transport may also contribute to the effects of lead on peripheral nerve conduction velocity. Landrigan et al. (1976) have noted a significant correlation between blood lead and decreasing nerve conduction velocity in children in a smelter community. This effect may indicate advancing peripheral neuropathy.

Paralleling these cellular or biochemical effects are electrophysiological changes indicating the perturbation of peripheral and central nervous system functioning observed in children with blood lead levels of 15 ug\dl and even below. These include slowed nerve conduction velocities (Landrigan et al., 1976), reaction-time and reaction-behavior deficits (Winneke et al., 1984;

Yule, 1984), as well as persistent abnormal EEG patterns including altered brain stem and auditory evoked potentials down to 15 ug/dl (Benignus et al., 1981; Otto et al., 1981, 1982, 1984). Neurological effects of lead at such low levels are particularly important because two- and five-year follow-up studies (Otto et al., 1982, 1984) indicated some persistent effects.

A recent article (Schwartz and Otto, 1987) has confirmed earlier findings of hearing effects related to low and moderate lead exposure. This study also found lead levels significantly related to major neurological milestones in early childhood development, including the age at which a child first sat up, walked or spoke. No threshold was evident for either the hearing or developmental effects.

Animal studies have also noted aberrant learning behavior at lower blood-lead levels. Crofton et al. (1980) found that the development of exploratory behavior by rat pups exposed to lead in utero lagged behind that of control rats. Average blood-lead levels on the 21st postnatal day were 14.5 ug/dl* for the exposed pups and 4.8 ug/dl* for the controls.

Gross-Selbeck and Gross-Selbeck (1981) found alterations in the operant behavior of adult rats after prenatal exposure to lead via mothers whose blood lead levels averaged 20.5 ug/dl.* At the time of testing (3 to 4 months, postnatal), the lead-exposed animals' blood-lead levels averaged 4.55 ug/dl* compared to 3.68 ug/dl* in the controls. This suggests that changes in central nervous system function may persist for months after the cessation

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* Blood lead levels in animals are not comparable to human blood-lead concentrations.

of exposure to relatively low blood-lead levels. In addition, animal studies show that behavior effects include both reduced performance on complex learning problems and signs of hyperactivity and excessive response to negative reinforcement (Winneke, 1977 and 1982a).

Finally, these effects show signs of a dose-response relationship. In children with high level lead poisoning, neurological damage is indisputable and mental retardation is a common outcome. For children with somewhat lower blood-lead levels, de la Burde and Choate (1972, 1975) found lesser but still significant cognitive effects, including lower mean IQs and reduced attention spans. Several studies have found smaller effects at lower blood-lead levels. Some very recent studies have also shown previously-undetected, significant cognitive effects in poor black children in the normal range of blood lead levels (from 6 ug/dl) without exhibiting an evident threshold.

While some of these effects have only been observed at higher blood-lead levels, in animals, or in vitro, they show a consistent dose-dependent interference with normal neurological functioning. Furthermore, several of these effects have been documented to occur at very low blood-lead levels (< 10 ug/dl) in children, with no clear threshold yet evident.

III.B.2.a. Cognitive Effects of Lower Blood-Lead Levels

The earliest study of cognitive effects from relatively low levels of lead exposure was done by Needleman et al. (1979), using shed deciduous teeth from over 2,000 children to index lead exposure. Among other findings, this study showed evidence of a

continuous dose-response function relating lead levels to behavior as rated by the teacher in terms of attention disorders. The authors also divided the subjects into a high- and low-lead group; significant effects ($p < 0.05$) were reported for various IQ indices, for classroom behavior, and for several experimental measures of perceptual-motor ability. Numerous papers by Needleman and his co-workers have provided additional analyses and follow-up studies related to the original data. These are listed in the bibliography of this document and are summarized in the Criteria Document (1986; p. 12-85 ff).

There were many questions relating to the Needleman analysis. An Expert Committee on Pediatric Neurobehavioral Evaluations, convened by EPA, noted some methodological problems and asked for a reanalysis and some additional analyses (Expert Committee, 1983). Reanalyses were conducted by Needleman (1984), Needleman et al. (1985) and EPA. All of the reanalyses confirmed the published findings of significant associations between lead levels and IQ decrements. After controlling for confounding variables, the Needleman data show evidence of a 4 IQ-point decrement associated with blood levels of 30-50 ug/dl. This finding is consistent with earlier studies showing IQ decrements of 5 points or higher in children with blood lead levels $\geq$ 50 ug/dl discussed previously (de la Burde and Choate, 1972 and 1975; Rummo, 1974; Rummo et al., 1979; etc.).

Since Needleman's original study in 1979, many other analyses of cognitive effects related to low and moderate lead exposure have been published. While some of these studies, like the earlier

ones, suffer from some methodological flaws and the relatively small sample sizes in many of them made it difficult to prove a statistically significant effect,* the combined weight of the studies point to "various types of neural dysfunction in apparently asymptomatic children across a broad range of blood lead levels" (Criteria Document, 1986; p. 12-156), including small IQ decrements in children with blood lead levels of 15-30 ug/dl.

In addition, new studies have examined neurotoxic effects in younger children and infants. Section A (above) discussed results from studies that found an inverse association between blood lead levels and gestational age at blood lead levels found commonly in the general population. Because gestational age can affect mental development in infants, whatever mechanism lies behind that effect must be factored into the discussion of lead's neurological effects.

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* A statistical method for combining comparable studies to overcome the problem of small sample size is described in The Costs and Benefits of Reducing Lead in Gasoline (EPA, 1985 p. IV-33 ff.

For use in public policy making, rejecting the results of studies simply because they fail to attain significance at the 5 percent level may be inappropriate for two reasons. First, policy makers need to be concerned about both type I and type II errors. Significance tests guard only against the first type (falsely rejecting the null hypothesis of no effect); they help ensure that a regulation is not imposed when there is no adverse effect. Type II errors (failing to reject the null hypothesis when it is false) also can be costly, however, because they can result in the underregulation of a real hazard. With small sample sizes and subtle effects, the probability of a type II error can be large.

The second reason for caution in rejecting the results of these studies, particularly in this case, is that while several fail to attain statistical significance individually, they do show a consistent pattern: the children in the higher lead groups generally have lower mean IQs.

Several studies assessed the relationship between blood lead levels at birth and the subsequent mental development of the infants. Bellinger et al. (1984), observing 216 middle- and upper-middle class children aged 6 months to 2 years in a prospective longitudinal study of early developmental effects of lead, found scores on the Bayley Mental Development Index (MDI) inversely related to umbilical cord blood-lead levels. The subjects were divided into three groups with mean blood-lead levels of 1.8 ug/dl (the low group), 6.5 ug/dl (midgroup), and 14.6 ug/dl (high). Gestational age and some other variables were identified as confounders of the association between cord blood lead and the MDI;* these confounding (positive) associations reduce the degree of association between cord blood lead and the MDI. Adjusting for confounding, and controlling for all known relevant factors, the difference in scores between the high and low blood-lead level groups was about 6 points on the MDI. Follow-up studies (Bellinger et al., 1985; 1986a; 1986b) indicated that the association between cord blood-lead level and MDI score continued for at least two years; no association was found with post-natal blood-lead level.

Vimpani et al. (1985), in a longitudinal study of almost 600 children at age 24 months, also found a statistically significant relationship between blood lead levels in infants and their performance on the MDI. Ernhart et al. (1985 and 1986) investigated prenatal lead exposure and post-natal neurobehavioral function,

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* That is because gestational age is also related to cord blood lead levels.

as well. Of the 17 neurobehavioral measures examined in the Ernhart studies, three showed significant relationships to blood lead levels.

Interim results of a longitudinal study presented by Dietrich et al. (1986), observing inner-city children in Cincinnati, showed evidence of an inverse relation between blood lead levels at three months with performance on three major mental development indexes, including the MDI. But these interim findings showed an association only for white infants. This and other analyses have also shown indirect effects on mental development and performance through lead's effect on gestational age and/or birth weight (cf. Addendum to the Criteria Document, 1986; p. A-35f).

In addition, several studies show an association between blood lead levels and other neurobehavioral patterns. Ernhart (1985 and 1986) showed that prenatal lead exposure correlated with certain neonatal behavior such as jitteriness and hyper-sensitivity, as measured on the Neurological Soft Signs scale. A follow-up study (Wolf et al., 1985) showed evidence that lowered Bayley MDI scores for one year olds was a sequela of the cord blood-lead relation shown on the Neurological Soft Signs scale after birth. And Winneke et al. (1985a) showed a significant relationship between perinatal blood-lead levels and one measure of psycho-motor ability at ages 6-7.

Finally, two new general population studies (Schroeder et al., 1985; and Schroeder and Hawk, 1986) investigated low socio-economic status children with blood lead levels in line with (or just slightly higher than) levels in the general population, controlling

for socio-economic factors, age, race, etc. The first study examined 104 lower SES children with blood lead levels ranging from 6-59 ug/dl (mean about 30 ug/dl). This study found a significant effect ($p < 0.01$) of lead upon IQ, which was sufficient to disrupt the normal mother-child IQ correlation. The second study, replicating the previous study with 75 low SES black children showed a highly significant relationship ($p < 0.0008$) between IQ and blood lead levels over the low to moderate range of 6-47 ug/dl. These studies suggest that lower socio-economic status places children at greater risk of the deleterious effects of low-level lead exposure on cognitive ability, while confirming that other factors (maternal IQ, home environment, etc.) are also closely related to IQ.

Winneke et al. (1985a and 1985b) also examined the predictive value of different markers of lead exposure for subsequent neurobehavioral development. Of an original study of 383 children at birth, 114 subjects were followed-up at ages 6-7. Mean blood-lead levels (maternal and infant) had been 8-9 ug/dl (range: 4-31 ug/dl). Regression analyses showed that maternal blood-lead levels (related closely to umbilical cord levels) accounted for nearly as much of the variance in neurobehavioral test scores at age 6-7 as did contemporary blood-lead levels.

The combined results of available studies of cognitive effects at low and moderate lead levels present evidence of potential IQ decrements and other cognitive impacts due to lead exposure at blood lead levels found commonly in the U.S. population; i.e., at 15 ug/dl and below, and possibly as low as 6 ug/dl for some groups of children.

III.B.3. The Magnitude of Lead's Impact on IQ

The studies summarized above indicate that among the cognitive effects resulting from exposure to lead is a potential lowering of children's IQs and a reduction in their ability to perform well in school. The latest draft of the Criteria Document (1986) characterizes the evidence as suggesting that, on average, blood lead levels of 50 to 70 ug/dl could correlate with average IQ decrements of five points, blood lead levels of 30 to 50 ug/dl could be associated with a four-point decrement in IQ, and that lead levels of 15 to 30 ug/dl could be related to IQ reductions of one-two points (p. 12-156, 12-282, and elsewhere). In Section III.D, we monetize the benefits of reducing these effects using the costs of compensatory education and potential decreases in future earnings resulting from decreased IQ.

These levels of effects may be associated with relatively consistent and/or relatively long exposure periods, possibly even several years. Permanent IQ effects may result only from fairly long periods of exposure, and a child who has a certain blood lead level for a relatively short amount of time (perhaps, a few months) may not suffer the full effect. Because of this uncertainty, for the effect upon decreased future earnings, we assumed conservatively that a child must be at a certain lead level for 3-4 years before permanent and irreversible IQ loss results.

This is an extremely conservative assumption. The data on prenatal exposure (obviously limited to at most nine months) show significant effects (both physical and neurological) persisting for two years or more. Fetal development is arguably particularly

vulnerable to disruption. Nonetheless, much data suggest that post-natal exposure periods of a year or so produce detectable effects. In addition, even if a lead-induced lag in cognitive or physical development were no longer detectable at a later age, this does not necessarily mean that the earlier impairment was without consequence. Given the complex interactions that contribute to the cognitive, emotional, and social development of children, compensations in one area of a child's development may exact a cost in another area. Unfortunately, very little is known about how to accurately measure these interdependencies. We have chosen the conservative estimate of 3-4 years of exposure because of unanswered questions of reversibility and permanence.

The approach of ascribing benefits only to those children who are brought below a critical threshold (15 ug/dl, 30 ug/dl or 50 ug/dl) by this proposed rule suffers from several faults, which cut in opposite directions and may offset each other. Categorization does not account for the fact that some children who are prevented by the regulation from going over a given threshold will do so by a narrow margin (e.g., their blood lead level will be 29 ug/dl when it would have been 31 ug/dl in the absence of the rule); such children are unlikely to receive the full four-point gain in IQ but they will receive more than one point. This means that the benefits of this potential rule may be overestimated.

On the other hand, categorization attributes no benefit to children whose blood lead levels are reduced from very high levels,

but not brought below a given threshold or to those whose levels would have been between two thresholds without the rule, but whose levels decrease further by the reduction in lead in drinking water. Also, it is quite possible that children suffer long-lasting, even permanent, effects with shorter exposure periods than the 3-4 years we assumed. These factors indicate that our benefit estimate may be too low.

III.C. Fetal Effects

Lead's adverse effects upon human reproductive functions have been known for over 100 years.* In 1860, for instance, Paul published findings (cited in the Criteria Document, p. 12-192) that lead-poisoned women were likely to abort or deliver stillborn infants. Because lead passes the placental barrier and fetal lead uptake continues throughout development, a growing concern in the public health community is that the most sensitive population for lead exposure is fetuses and newborn infants. This concern is supported by both animal and human studies.

Several categories of fetal effects were discussed previously. Within Section A, above, in the discussion of lead's adverse effect upon children's physical growth and development we presented data on the inverse relationships between blood lead levels and gestational age, birth weight and birth height. Section B, also above, describes the neurotoxic effects of lead, including the inverse relationship between blood lead level and

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* Indeed, 'lead plasters' were used as abortifacients at the turn of the century.

infant mental development as measured by several different neurological indices.

In addition, several studies have implicated lead in complications of pregnancy, including early and still births, and, possibly, low-level congenital anomalies. (Lead's adverse effects upon reproductive function are discussed in Section IV.B. of the next chapter.) As discussed previously, lead has a negative effect upon gestational age. As early examples of these findings, Fahim et al. (1976) found that women who had normal full-term pregnancies had average blood-lead levels of 14.3 ug/dl, whereas women with early membrane rupture had average blood-lead levels of 25.6 ug/dl, and women with premature delivery had average blood-lead levels of 29.1 ug/dl. Wibberly et al. (1977) found that higher lead levels in placental tissues were associated with various negative pregnancy outcomes, including prematurity, birth malformation, and neonatal death. Bryce-Smith et al. (1977) found bone lead concentrations in still births of 0.4-24.2 parts per million (ppm) in the rib (average: 5.7) versus typical infant bone lead levels of 0.2-0.6 ppm.

Needleman et al. (1984) analyzed data from over 4,000 live births at Boston Women's Hospital and reported an association between minor congenital anomalies and umbilical-cord blood-lead levels. There was no association between any particular malformation and lead, but only between all minor malformations and lead. There also were no significant associations between lead and any major malformations, although given the rate of such malformations in the general population, a sample this size has little power to detect such an effect. Holding other covariates

constant, the relative risk of a child demonstrating a minor malformation at birth increased by 50 percent as lead levels increased from 0.7 ug/dl to 6.3 ug\dl (the mean cord-lead level). This risk increased an additional 50 percent at 24 ug/dl. (Umbilical-cord blood-lead levels are generally somewhat lower than, but correspond to, maternal blood-lead levels; e.g., Lauwerys et al., 1978.)

Two other studies (McMichael et al., 1986; Ernhart et al., 1985 and 1986) investigated the association between pre-natal lead exposure and congenital morphological anomalies. They did not find a similar occurrence of congenital anomalies. on the other hand, the Needleman analysis relies upon a much-larger data base than either the Ernhart or McMichael studies. Nonetheless, the available evidence on lead's effect on congenital anomalies allows no definitive conclusion about low-level lead exposures and the occurrence of congenital anomalies.

Finally, Erickson et al. (1983) found lung- and bone-lead levels in children who died from Sudden Infant Death Syndrome were significantly higher ($p < 0.05$) than in children who died of other causes, after controlling for age. While this study suggests a potential relationship between lead exposure and Sudden Infant Death Syndrome, this issue also remains to be more fully evaluated.

III.C.1. Assessing the Benefits of Reduced Fetal Exposure to Lead

Lead crosses the placental barrier and fetal uptake of lead continues throughout development. Fetal and new-born blood-lead

levels are closely related to, though generally slightly lower than, maternal levels.* In addition, during physiological conditions of bone dimineralization, which are known to occur during pregnancy and lactation, lead as well as calcium may be released from its storage in bone. A readily mobile compartment of skeletal lead has been demonstrated in humans (Rabinowitz et al., 1977) and in experimental studies both in vivo (Keller and Doherty, 1980a and 1980b) and in vitro (Rosen, 1983; Pounds and Rosen, 1986). For these reasons, to assess the benefits of EPA'S proposed reduction in the MCL for lead, we are concerned with two populations: pregnant women currently at risk of receiving high levels of lead in their drinking water and especially those women (aged 15-44) who are likely to have blood lead levels that could present a risk to the unborn child.

To determine what blood lead level should be used as a cut-off for estimating risk to the fetus from lead exposure, two recent policy actions related to EPA rules were considered. In the Spring of 1985, EPA's Clean Air Science Advisory Committee recommended a goal of preventing children's blood-lead levels

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* "Exposure levels during the course of pregnancy may not be accurately indexed by blood lead levels at parturition. Various studies indicate that average maternal blood-lead levels during pregnancy may tend to decline, increase, or show no consistent trend. These divergent results may simply reflect the likelihood that the maternal blood lead pool is subject to increase as bone stores of lead are mobilized during pregnancy and to decrease as lead is transferred to the placenta and fetus. Apparently, then, under some conditions the fetus may be exposed to higher levels of lead than indicated by the mother's blood lead concentration." (Text quoted from The Addendum to the Criteria Document, 1986; p. A-45. See references cited there.)

from exceeding 15-20 ug/dl. This goal was supported by the epidemiological and toxicological data at the time that showed significant lead-induced health effects beginning to be detectable in that range. More recently, the Addendum to the Criteria Document (US-EPA, 1986; p. A-48) stated, "At present, . . . , perinatal blood lead levels at least as low as 10 to 15 ug/dl clearly warrant concern for deleterious effects on early postnatal as well as prenatal development."

To be conservative, we used the intersection of these two ranges -- 15 ug/dl -- as the blood lead level of concern for fetal effects. As the simplest relationship between maternal and fetal blood-lead levels, we have assumed equivalence. This ignores some findings that the rate of fetal absorption of lead may increase throughout development (e.g., Barltrop, 1969; Rabinowitz and Needleman, 1982; Donald et al., 1986). (See the Criteria Document, Sections 10.2.4 and 12.6, for a fuller discussion of this issue.)

The Air Quality Criteria Document estimates that there are approximately 54 million women of childbearing age (i.e., between 15 and 44 years old), of whom about 7 percent are likely to be pregnant at any given time (1986; p. 13-47); this is about the same percentage as the annual birth rate given by the Census Bureau: 67.4 per 1,000 women aged 15-44.* Census Bureau data (presented in Current Population Reports, Series P-25, or summarized in Table 27 in Statistical Abstracts, 1986) show that 24 percent

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* 1985 Statistical Abstracts of the United States (1986), Table 82.

of the total population is women aged 15-44. Assuming that women of childbearing age and that pregnant women are distributed proportionately among those served by community water systems and by private or non-community water supplies, and assuming that these women are distributed proportionately between community water systems with high and low lead levels, these figures yield the following estimates of fetuses at risk of exposure to lead levels exceeding 20 ug/l in drinking water.

24% x 42 million* x 67.4 per thousand = 680,000 fetuses at risk

Calculating the number of women at high blood-lead levels is more difficult. Our population models do not yet include sufficient data on all adult women in the United States because our analyses so far have focused, separately, on children and adult men. However, some data are available that enable us to make very crude exposure estimates for this population.

The Hispanic Health and Nutrition Examination Survey (called either the Hispanic HANES or HHANES), conducted by the National Center for Health Statistics (NCHS) between 1982 and 1984 contains relatively recent data on blood lead levels in the U.S., including adults. These data, published and available from NCHS, indicate that in 1988 Mexican American women aged 15-44 will have an estimated mean blood-lead level of 7.1 ug/dl, with a geometric standard deviation of 1.5; they also show that an estimated 0.36

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* Estimate of people in the United States served by community water supplies who currently receive water that exceeds 20 ug/l. Methodology presented in Chapter II.

percent of those women will have blood lead levels over 15 ug/dl in 1988.* While we do not know how data on Mexican Americans compare to data on white Americans, these estimates certainly significantly underestimate the lead levels of black Americans, which are generally much higher than whites.** Assuming that all women in the U.S. have blood lead levels and distributions comparable to those in the Hispanic HANES:

$$54 \text{ million} \times 0.36\% \times \frac{219}{240} \text{ million} = 177{,}000$$

women in 1988 served by community water supplies are likely to have blood lead levels over 15 ug/dl, of whom

$$177{,}000 \times 7\% = 12{,}400$$

are likely to be pregnant in any given year. For these women, any contribution of lead from drinking water is a potential health risk to the fetus because they are already at the cut-off point recommended by the Clean Air Science Advisory Committee, and the fetus may take the bulk of the lead absorbed by the mother.

Determining what fraction, if any, of these most-at-risk fetuses (i.e., of mothers with blood lead levels > 15 ug/dl and receiving water > 20 ug/l) is included in the 680,000 at-risk

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* These estimates rely upon the coefficients on the distribution of blood lead levels in the country from the NHANES II (discussed previously) and Census Bureau data on the demographics of the population. These extrapolations assume reduced exposure to lead from gasoline.

** For an indication of the racial differences in the distribution of blood lead levels, look at the data presented on Table III-1, on p. III-8 above.

fetuses (i.e., of mothers receiving water > 20 ug/l) estimated above is difficult. To avoid double-counting we have used the most conservative and least controversial assumption: that all of the most-at-risk fetuses are included in the 680,000 fetuses exposed in utero to drinking water exceeding the proposed MCL. Therefore, the proposed MCL would protect 680,000 fetuses in 1988.

III.D. Monetized Estimates of Children's Health Benefits

The health benefits of reducing children's exposure to lead are diverse and difficult to estimate quantitatively or to value in monetary terms. The monetized benefits include only two admittedly incomplete measures: savings in expenditures for medical testing and treatment, and savings associated with decreased cognitive ability. These measures of benefit exclude many important factors, such as the reproductive and stature effects discussed above. These and other limitations are discussed in Section III.E, below.

In fact, many children with elevated blood-lead levels are neither detected nor treated. However, this estimation procedure assumes that children who go undetected and untreated bear a burden at least as great as the cost of testing and providing the treatment (medical or educational) of those who are detected. So, all children with high blood-lead levels are assumed to incur "costs", whether medical expenditure costs or personal costs in the form of poor health, inadequate learning, decreased future vocational options, etc.

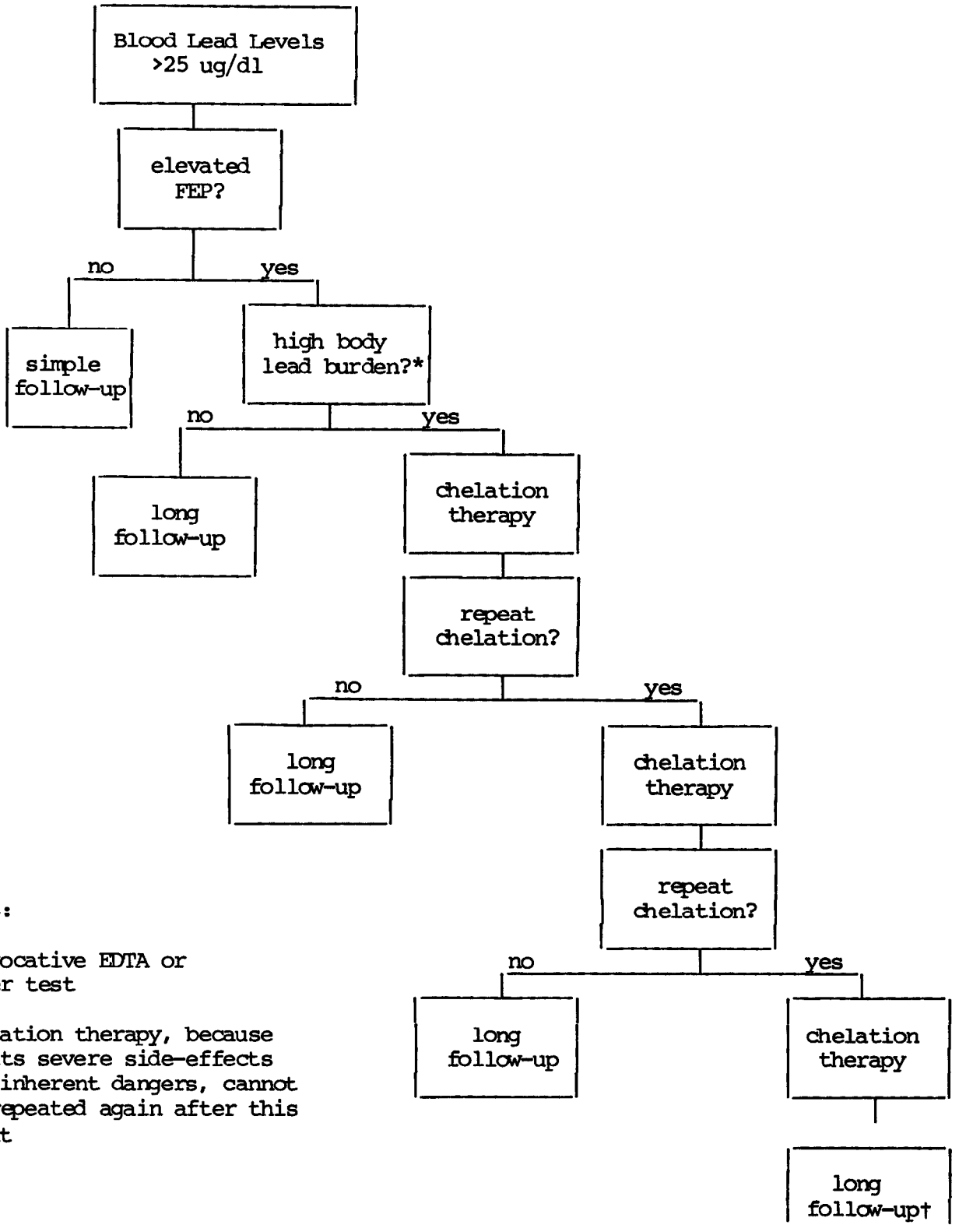
III.D.1 Reduced Medical Costs

To estimate the benefits of reduced medical care expenses, we used the optimal diagnosis, treatment, and follow-up protocols recommended by Piomelli, Rosen, Chisolm, and Graef in the Journal of Pediatrics (1984). Piomelli et al. estimated the percentages of children at different blood-lead levels who would require various types of treatment. Figure III-4 summarizes the treatment options that we used, based on the recommendations of Piomelli et al.

An evaluation of typical medical services suggests that administrative expenditures and follow-up tests would cost $110 (1985 dollars) for each child found to be over 25 ug/dl at screen ing. Of those children over 25 ug/dl blood lead, based on Piomelli et al. (1982) and Mahaffey et al. (1982), we estimated that 70 percent would be over 35 ug/dl EP. Piomelli et al. (1984) recommend provocative ethylenediamine-tetraacetic acid (EDTA) testing for such children. EDTA testing typically requires a day in the hospital and a physician's visit; based upon hospital cost data, we assessed a cost of $540 per test (1985 dollars). We also assumed that all children receiving EDTA testing would receive a series of follow-up tests and physicians' visits, costing an estimated $330 (1985 dollars).

The purpose of EDTA testing is to see if children have a dangerously high body-lead burden (a lead excretion ratio over 0.60, per Piomelli et al.). Table III-2 presents Piomelli et al.'s estimates of the percentages of children at various blood lead levels who will require chelation therapy; it ranges from a

FIGURE III-5. Flow Diagram of Medical Protocols for Children with Blood Lead Levels above 25 ug/dl



NOTES:

*Provocative EDTA or other test

†Chelation therapy, because of its severe side-effects and inherent dangers, cannot be repeated again after this point

TABLE III-2. Percent of Children Requiring Chelation Therapy

Blood Lead Levels	Percent
25-30 ug/dl	0*
30-39 ug/dl	
age three and over	9.6*
age under three	11.5*
40-49 ug/dl	
age three and over	26.0
age under three	37.9
50-59 ug/dl	
age three and over	36.0
age under three	49.0
above 59 ug/dl	100.0

Source: Piomelli et al., 1984

* At blood lead concentrations of 25 to 35 ug/dl, 6-7 percent of children require chelation therapy (Piomelli et al., 1984). The presentation above, while consistent with the data and other presentations, can easily result in an underestimation of risks.

In addition, given the degree of non-linearity between blood lead levels and chelatable lead, it is possible that some children between 15 and 24 ug/dl (a concentration below CDC'S definition of 'elevated' blood lead) may have positive EDTA test results. Thus, the percentages above may represent conservative estimates. (Based upon data and discussions with Dr. John Rosen, Department of Pediatrics, Montefiore Medical Center, Albert Einstein College of Medicine; one of the authors of the Piomelli article.)

low of zero for those under 30 ug/dl to a high of 100 percent for those over 59 ug/dl.

Based on the data from the NHANES II, we estimated that, of those children over 25 ug/dl blood lead, about 20 percent are between 30 and 40 ug/dl and 10 percent are over 40 ug/dl. Using those estimates and the percentages in Table III-2, 5 percent of the children above 25 ug/dl would require chelation therapy. In addition, we estimated that half of those children chelated would require a second chelation due to a rebound in their blood lead level, and that half of those children would require a third chelation treatment. Thus, a total of 0.0875 chelations would be required for every child over 25 ug/dl blood lead at screening. To calculate the total cost per chelation, we estimated that it would require five days in the hospital, several physicians' visits, laboratory work, and a neuropsychological evaluation, for a total cost of about $2,700 per chelation.

Multiplying each of these costs by its associated probability and then summing them yields the estimated cost per child over 25 ug/dl:

$$1.0(\$110) + 0.7(\$540) + 0.7(\$330) + 0.0875(\$2,700) = \$955.25,$$

which we round to $950 in 1985 dollars.

Because we have not included welfare losses (such as work time lost by parents), the adverse health effects of chelation therapy itself (such as the removal of necessary minerals and potential severe kidney damage), or such non-quantifiables as the

pain from the treatment, this estimate of the benefits is conservative. As mentioned previously, these medical costs are a measure of avoidable damage for all the incremental cases of lead toxicity, whether detected or not.

III.D.2. Costs Associated with Cognitive Damage

The studies on the neurotoxicity of lead show a continuum of effects, in a dose-response relationship, from low to high levels of exposure. Manifestations of this neurotoxicity are varied and include IQ deficits and other cognitive effects, hearing decrements, behavioral problems, learning disorders, and slowed neurological development. Several of lead's neurotoxic effects can combine, and a few studies (e.g., de la Burde and Choate, 1972, 1975) show poorer performance in school associated with higher blood-lead levels. For instance, these studies showed that children with higher blood-lead levels were seven times more likely than similar children with lower lead levels to repeat a grade, to be referred for psychological counseling, or to show other signs of significant behavioral effect. Supplementary educational programs may compensate for some of these effects, though certainly not all of them.

Because of the difficulties inherent in monetizing neurological effects, we selected only one sub-category to investigate further -- cognitive damage resulting from exposure to lead. We developed two methods for calculating the benefits of reduced cognitive damage: compensatory education costs (as a proxy for the damage) and decreased future earnings as a function of IQ points lost.

III.D.2.a. Compensatory Education

To estimate roughly the cost of compensatory education for children suffering low-level cognitive damage, we used data from a study prepared for the Department of Education's Office of Special Education Programs. Kakalik et al. (1981) estimated that part-time special education for children who remained in regular classrooms cost $3,064 extra per child per year in 1978; adjusting for changes in the GNP price deflator yields an estimate of $4,640 in 1985 dollars. This figure is quite close to Provenzano's (1980) estimate of the special education costs for non-retarded, lead exposed children.

In developing the algorithm for calculating a unit cost for compensatory education, we made three relatively conservative assumptions. First, we assumed that no children with blood lead levels below 25 ug/dl would require it. This is conservative because many studies show detectable cognitive effects at 15 ug/dl. Second, we assumed that only 20 percent of the children above 25 ug/dl would be severely enough affected to require and receive some compensatory education. Third, based upon several follow-up studies that showed cognitive damage persists for three years or more (even after blood lead levels have been lowered), we assumed that each child who needed compensatory education would require it for three years but that the damage would then be compensated for. This is conservative for two reasons. As a neurotoxin, lead affects many capacities: hearing, motor coordination and other sensory perceptions, as well as cognitive abilities. No part-time in class special education can possibly compensate for

all these effects. In addition, while no studies have yet been published on this, data from several large lead poisoning prevention programs (records on about 1,600 children treated at the Montefiore Center in New York) show that once lead toxic children require compensatory education, such educational services generally will be required for many more than three years. We have used the three-year cut-off because the only published follow-up studies show cognitive effects persisting for (at least) that period of time. Thus, the estimated average annual cost per child over 25 ug/dl is

$$(0.20) \times (3) \times (\$4,640) = \$2,784,$$

which we round to $2,800 (1985 dollars), for compensatory education to address lead's cognitive damage.

III.D.2.b. Effect Upon Future Earnings

Literature concerning the economic returns of schooling has included some investigation of the impact of IQ upon earnings; a survey of this literature was prepared for EPA by ICF (ICF, 1984) and peer reviewed by a panel of distinguished economists. Typically, estimates of the returns to schooling are based upon an "earnings capacity" that consists of equations for schooling, "ability" (usually measured by scores on standardized IQ tests), and socioeconomic variables. Both the main subject (economic returns of schooling) and its off-shoot (earnings as a function of IQ) are extremely complicated and controversial.

Despite the wide variety of data sets and methodologies used to examine these issues, the estimates of the direct effect of

ability on earnings appear to be fairly consistent. With one exception, estimates of the direct effects of a one point change in IQ fall between 0.20 and 0.75 percent of future expected earnings. There are fewer estimates of the indirect effects of IQ on future earnings (which include the impact of IQ on the schooling of the child, which in turn affects expected earnings) and they range from 0.18 to 0.56 percent per IQ point. Combined, a one IQ-point change is associated with a change of 0.65 to 1.15 percent in earnings. We used the arithmetic mean (one IQ point = 0.90 percent of earnings) to calculate the benefit of this rule.

As summarized above (Section III.B.), the literature indicates that children with blood lead levels between 15 and 30 ug/dl could suffer IQ losses of 1-2 points (for which we used the arithmetic mean -- 1.5 points -- as the point estimate), between 30 and 50 ug/dl children could lose 4 IQ points, and over 50 ug/dl they could lose 5 IQ points. Because permanent IQ damage probably occurs after a year or more of lead exposure, we assumed conservatively that children would suffer these losses after 3-4 years of exposure at these levels.* To calculate the annual benefits of this proposed rule, therefore, the potential effect upon future earnings resulting from these exposure levels was divided by 3.5 years. Multiplied together, reducing a child's blood-lead level below 15 ug/dl could increase expected future lifetime earnings by 0.4 percent

(0.9% x 1.5 IQ points ÷ 3.5 years = 0.4%),

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* Because recent data is showing that much shorter exposure periods produce effects that can last for at least 2-3 years, subsequent analyses will have to re-examine this assumption.

reducing a child below 30 ug/dl could increase earnings by 1.0 percent

(0.9% x 4 IQ points ÷ 3.5 years = 1.0%),

and reducing a child below 50 ug/dl could increase earnings by 1.3 percent

(0.9% x 5 IQ points ÷ 3.5 years = 1.3%).

The present value of expected lifetime earnings is $687,150;* deferred for 20 years at 5 percent real discount rate** reduces it to $258,950 (1985 dollars). The effect of the cognitive damage would decrease expected future earnings by $1,040 (1985 dollars) for a child brought below 15 ug/dl; $2,600 (1985 dollars) for a child brought below 30 ug/dl; and $3,350 (1985 dollars) for a child brought below 50 ug/dl.

To calculate the annual benefits in this category, the number of children who would be brought below each of these points (15, 30 and 50 ug/dl) was multiplied by the change in expected future lifetime earnings. Table III-3 presents those benefit calculations for the proposed MCL reduction to 20 uEarnings for sample year 1988.

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* Calculated from Bureau of the Census data: Lifetime Earnings Estimates for Men and Women in The United States: 1979 (1983) --p.3 -- and 1985 Statistical Abstracts of the United States (1986) -- Table 216. Converted to 1985 dollars.

** These costs are deferred because those suffering the effects are children and will not enter the work force for up to 20 years. Obviously, using the largest deferral period (20 years) reduces the value of the benefit and reduces the benefit estimate, whereas 8- or 10-year-old children may begin working within 8 years and so would have a much shorter deferral period. This biases the estimates downward slightly.

TABLE III-3. Estimated Annual Benefits of Reduced IQ Damage by Using Changes in Expected Future Lifetime Earnings, For Sample Year 1988

	Blood Lead Level			
	15 ug/dl	30 ug/dl	50 ug/dl	TOTAL
Number of children	230,000	11,000	100	241,100
IQ points potentially lost	1-2 per child	4 per child	5 per child	NA
Present value of decreased earnings (1985 dollars)	$1,040 per child	$2,600 per child	$3,350 per child	NA
TOTAL (1985 dollars)	$239.2 million	$28.6 million	$ 0.3 million	$268.1 million

III.D.3. Summary of Monetized Benefits

Table III-4 summarizes the estimates of the monetized annual children's health benefits of the potential rule for one sample year: 1988. Adding the estimates of compensatory education ($2,800) and medical costs ($950) yields a combined annual benefit estimate of $3,750 per case avoided of a child's blood-lead level exceeding 25 ug/dl. This is Method 1 in Table III-4. Method 2 is medical expenses plus decreased future earnings as a consequence of lead's adverse effect upon IQ. The benefits of avoided cognitive damage calculated as a function of IQ's relationship to expected future earnings are not linear, however; they are a step function.* Therefore, average costs per child were not calculated. Instead, we calculated the annualized benefit from avoided IQ losses of reducing 230,000 children below 15 ug/dl; 11,000 children below 30 ug/dl; and 100 below 50 ug/dl, and combined that with the total medical expenses avoided for bringing 29,000 children below 25 ug/dl. Note that the difference between Method 1 and Method 2 is that they include alternative methods for valuing aspects of the cognitive damage resulting from exposure to lead.**

The benefits for Method 1 (medical costs plus compensatory education) are not absolutely comparable to those from Method 2, for three reasons. First, Method 1 -- based upon per child estimates -- is strictly a function of the number of children

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* Measurements were taken for the step function at 15 ug/dl, 30 ug/dl, and 50 ug/dl.

** This biases the results downward because there is a strong rationale for considering these effects as additive. Compensatory education is unlikely to fully compensate for the neurological damage caused by exposure to lead and so, effects upon future ability and performance could still result in decreased earnings.

TABLE III-4. Monetized Annual Benefits of Reducing Children's Exposure to Lead Using Alternative Methods, for Sample Year 1988 (1985 dollars)

	METHOD 1			METHOD 2		
	Medical Expenses	Compensatory Education Costs	Total Method 1	Medical Expenses	Earnings Lost	Total Method 2
Number of children	29,000	29,000	29,000	29,000	241,100	241,100
Costing unit	$950 per child	$2,800 per child	$3,750 per child	$950 per child	$268.1 million total	NA
Total benefits	$27.6 million	$81.2 million	$108.8 million	$27.6 million	$268.1 million	$259.7 million

passing one critical point: 25 ug/dl. Method 2, on the other hand, depends upon the distribution of children by blood lead levels, and how the distribution changes* as a result of EPA's proposed regulatory action. Second, Method 1 ascribes no benefit to any health effects below 25 ug/dl, while Method 2 includes neurotoxic effects between 15 and 25 ug/dl. Finally, Method 2 measures effects to a child over a working life for each year of exposure, while Method 1 includes only costs incurred for those children who receive compensatory education for the duration of that benefit.

In addition, while they are discussed qualitatively, no monetary value is assigned to the fetal effects, the increased risk of anemia, metabolic changes or the negative impact upon stature. So the monetized benefit estimates omit many important categories, and thus are likely to be significant underestimates of the total benefits of reducing lead in drinking water. The next section contains a discussion of some of these factors.

III.E. Valuing Health Effects: Caveats and Limitations

To begin valuing the health effects that would be avoided as a result of the new MCL for lead in drinking water, we estimated 1) medical treatment and monitoring costs for those children whose blood lead levels reach or exceed the criteria recommended by the Centers for Disease Control as determining lead toxicity

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* That is, how many children pass each of several critical points, depending upon the category: 15 ug/dl, 25 ug/dl, 30 ug/dl or 50 ug/dl.

(25 ug/dl of blood lead when combined with erythrocyte proto-porphyrin levels of 35 ug/dl), and 2) two alternative ways of valuing the cognitive damage resulting from lead exposure: the cost of part-time in-class compensatory instruction as a proxy for the cognitive damage that lead causes and decreased expected future earnings as a function of IQ loss.

The cost-of-illness estimates themselves are low, primarily because, to reduce potential controversy, the calculations rely upon many conservative assumptions. For instance, the monetization of compensatory education costs is based upon likely practice and not preferred treatment. Although children suffering cognitive damage from lead exposure should receive more intensive and extensive educational resources, the Department of Education estimated that they would probably receive only part-time in-class remedial/compensatory help. In addition, the estimate that only 20 percent of children over 25 ug/dl would receive any extra help is conservative. The real (social) cost of the illness does not decrease if not all victims receive the treatment they need; assuming the treatments are efficacious, children who are left with diminished cognitive abilities incur a cost at least equal to the cost of the treatment they should have (but did not) receive. The health benefit estimates, therefore, should be understood as very low lower-bounds for these categories of effects.

We have also not conducted cost-of-illness calculations for most of the adverse health effects associated with human exposure

to lead. Among the many effects not valued monetarily in the health benefits analysis are:

- kidney effects, detectable in children at about 10 ug/dl;
- hematopoietic damage, detectable in children at below 10 ug/dl;
- neurological effects in children below the level of lead toxicity, with central nervous system effects detectable at below 10 ug/dl and no perceived threshold;
- metabolic changes, detectable in children at about 12 ug/dl;
- enzymatic inhibition, with no threshold indicated in children, even below 10 ug/dl;
- all effects on fetuses in vitro, although lead crosses the placental barrier and maternal blood-lead levels correlate with adverse pregnancy outcomes, including decreased gestational age, slowed mental and physical development in neonates, potential low-level congenital anomalies and other adverse outcomes, including fetotoxicity at high levels;
- stature effects on children, which are dose-dependent with no threshold evident; and
- effects upon other organ systems, for instance, immune and gastrointestinal.

Finally, three serious phenomena of lead's adverse effect upon human health are not included. First, hematopoietic, metabolic, and enzymatic damages have cascading effects throughout the body, which are not adequately addressed. Second, many of the specific effects have long-lasting sequelae that are not included.

And last, there is a significantly greater chance of serious effects later in life, including renal failure and neurological disorders, even in individuals whose highest detected blood-lead level was below that associated with the most severe effects and who did not at the time show evidence of lead toxicity; this risk is not included in the analysis.

In addition to the categories of adverse health effects for which we have not yet been able to quantify benefits at all, the costs of the illnesses that are calculated greatly underestimate the real (social) benefits of preventing those effects, even for the health categories evaluated. The underestimates occur because of the exclusion of some categories of direct costs associated with those effects and the total exclusion of all indirect but related costs (e.g., work time lost by the parents of lead-poisoned children).

In general, society's willingness-to-pay to avoid a given adverse effect is many times greater than the cost of the illness itself, so cost-of-illness analyses inherently underestimate the benefits of avoiding the adverse effect.* Willingness-to-pay studies indicate that society is usually willing to pay two to ten times the cost of medical treatment, and that in specific circumstances society is willing to pay a hundred or a thousand times the cost of the illness itself in order to prevent its occurrence.

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* For instance, in general people would be willing to pay more than the price of two aspirins to avoid having a headache.

More specifically, in the cost-of-illness analyses included in this report, only the expenses that are directly related to an individual's medical treatment for the specific symptom being evaluated at the time the symptom occurs were included. So, for instance, no costs are ascribed for the possibility of adverse effects from the medical treatment or hospitalization itself, or for the possibility that the specific effect of lead may precipitate or aggravate other health effects (e.g., children with anemia are more susceptible to many infections). Related expenses, such as the travel costs to obtain medical services or the costs of making the home environments of children suffering from lead toxicity safe for them (i.e., altering their diet to compensate for their propensity to anemia, removing all lead-laden dust, etc.) were also excluded. Finally, no value was ascribed to the pain and suffering of those affected; this is an especially significant omission because, as examples, chelation therapy is extremely painful and having a child with lead poisoning or who is hospitalized can totally disrupt family life.

We have also omitted all the indirect but related costs of lead's adverse effect upon human health. These include work time lost by friends and relatives of the victims (including the parents of lead-poisoned children); medical research related to the prevention, detection, or treatment of the effects of exposure to lead; the development of new procedures to correct the damage resulting from lead exposure; decreased future earnings for those suffering cognitive damage (other than very young

children) or physical incapacitation (including behavioral disorders) from lead's adverse effects upon virtually every human system; and the like.

III.F. Summary of Annual Monetized and Non-monetized Children's Health Benefits

This chapter presents evidence of a variety of physiological effects associated with exposure to lead, ranging from relatively subtle biochemical changes to severe damage and even death at very high levels. Of these, only two categories of effects are monetized: costs of medical treatment for children with elevated blood-lead levels and costs associated with the cognitive damage resulting from lead's neurotoxicity. For the latter category, two alternative monetization techniques were presented: compensatory education as a proxy measure and decreased future earnings as as function of IQ points lost. In addition, the numbers of children at risk of several other pathophysiological and neurotoxic damage each year, including those at risk of stature decrements, at increased risk of anemia, total number of children at risk of IQ-point-loss, and fetuses exposed to potentially dangerous lead levels, were estimated. Table III-5 summarizes both the monetized and non-monetized benefits of reducing exposure to lead in drinking water for one sample year, 1988.

TABLE III-5. Summary of Annual Monetized and Non-monetized Children's Health Benefits of Reducing Lead in Drinking Water for Sample Year 1988

Annual Monetized Benefits (1985 dollars)	
° Medical costs	$27.6 million
° Cognitive damage costs:	
compensatory education (Method 1)	$81.2 million
decreased future earnings (Method 2)	$268.1 million
TOTAL Method 1	$108.8 million
Method 2	$295.7 million
Annual Non-monetized Benefits (children at risk of:)	
° Requiring medical treatment	29,000
° Loss of 1-2 IQ points	230,000
4 IQ pints	11,000
5 IQ points	100
° Requiring compensatory education	29,000
° Stature decrement	82,000
° Fetuses at risk	680,000
° Increased risk of hematological effects	82,000

CHAPTER IV

HEALTH BENEFITS OF REDUCING LEAD: ADULT ILLNESSES

Concerns about the health effects of ambient exposure to lead traditionally have focused on children. Although lead has a variety of adverse effects on the health of adults, most of these effects were believed to be a risk only at high blood-lead levels. Recently, many analyses -- still the subject of some controversy -- have shown a robust, continuous relationship between blood lead levels and blood pressure in men, confirming a relationship between lead exposure and blood pressure that has been discussed in the experimental toxicology (animal experiments) literature. That finding has important implications for the benefits of reducing lead in drinking water because high blood pressure, in turn, is linked to a variety of cardiovascular diseases.

Other recent human studies show deleterious effects of lead exposure upon fetal and post-natal growth and development, both mental and physical, that can be correlated with exposure to lead in utero. These studies are discussed in the previous chapter on children's health effects. In this chapter, studies of reproductive effects on both men and women are summarized briefly, although no attempt is made to value these effects monetarily.

This chapter contains four sections. Section A discusses the relationship between body lead levels and blood pressure in adult males, and includes studies of heart disease as related to water hardness. Section B discusses some reproductive effects of lead exposure. Other health effects of lead, such as kidney function, immune system function, and hematological effects, are not discussed

in this document. The monetized and non-monetized benefits are summarized in Section C and some caveats and limitations of this analysis are found in Section D.

A more complete discussion of the relationship between lead and blood pressure is included in the Addendum to the Air Quality Criteria Document for Lead (U.S. EPA, 1986; appended to Volume 1). The methods for valuing monetarily the cardiovascular effects were developed in support of EPA's most recent rule reducing the amount of lead permitted in leaded gasoline. These methods are presented more fully in The Costs and Benefits of Reducing Lead in Gasoline (U.S. EPA, 1985b).

IV.A. The Relationship between Blood Lead Levels and Blood Pressure

This section analyzes the statistical relationship between blood lead and blood pressure. The first part provides a brief overview of human studies relating blood lead levels to blood pressure and from there links those changes to cardiovascular disease rates. The second part of this section discusses potential mechanisms and animal data related to lead's effect upon blood pressure. The Addendum to the Criteria Document for Lead (U.S. EPA, 1986; p.A-1 to A-31) contains a much fuller analysis of this issue and serves as the basis for the summary contained here; it also includes a full bibliography. The third part of this section describes studies that have investigated the potential relationship between cardiovascular disease rates and water hardness, and the possible role of lead in contributing to cardiovascular disease when present in water of different hardness.

While extremely important, these findings are still the subject of some controversy. In addition, these results are limited in several ways. All of the monetized benefits are restricted to males aged 40 to 59, because lead is statistically correlated with blood pressure only in men, not women,* and because better data are available for that age range. In addition, most of these estimates cover only white males, because the existing studies have had insufficiently large samples of non-whites. For these reasons, the cardiovascular health benefit estimates contained in this section are likely to understate significantly the adult health benefits of reducing lead in drinking water. The most important omissions are older males and black males of all ages.

IV.A.1. Epidemiological Studies of Blood Lead Levels and Hypertension

Lead has long been associated with effects on blood pressure and the cardiovascular system, including a paper in the British Medical Journal by Lorimer in 1886 that found that higher blood-lead levels increased the risk of hypertension. Until recently, most of the studies focused only on hypertension and relatively high lead-exposure levels, and did not look for a continuous effect of lead on blood pressure. Others failed to find effects of lead on hypertension that were significant at the 95 percent

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* Many fewer studies have investigated the relationship between exposure to lead and blood pressure in women. However, in the large-scale general population studies, while blood lead was positively correlated with blood pressure in women, it was not statistically significant (at the 90 percent confidence level).

confidence level, although most of them did find a positive association. A stronger and

> "more consistent pattern of results has begun to emerge from recent investigations of the relationship between lead exposures and increases in blood pressure or hypertension"
>
> (Addendum to the Criteria Document, p. A-2)

throughout the range of measured blood-lead levels in various clinically-defined, occupationally-exposed, or general population groups.

IV.A.1.a. Occupational Studies

Kirkby and Gyntelberg (1985) evaluated the coronary risk profiles of 96 heavily-exposed lead smelter workers with those of non-occupationally exposed workers, matched for age, sex, height, weight, socioeconomic status, and alcohol and tobacco consumption. There were no significant differences in life style habits, as far as could be determined. Diastolic blood pressure was significantly elevated among the lead workers, as was the percentage of lead workers with ischemic electrocardiographic (ECG) changes and some other factors. On the other hand, systolic blood pressure and some other cardiovascular risk factors, e.g., angina pectoris, were not significantly different. Overall, the authors concluded that long-term lead workers have higher coronary risk profiles than a comparable referent group and that these findings may indicate a greater risk for major cardiovascular diseases, such as myocardial infarctions or strokes.

Another study of about 50 occupationally-exposed workers (de Kort et al., 1986) also showed blood pressure levels to be positively correlated with blood lead levels at near or below 60-70 ug/dl, after controlling for confounding variables.

IV.A.1.b. Observational Studies

Moreau et al. (1982) found a significant relationship ($p < 0.001$) between blood lead levels and a continuous measure of blood pressure in 431 French male civil servants after controlling for age, body mass index, smoking, and drinking. In this study, the correlation was highest in young subjects, and decreased with age. The effect was stronger for systolic pressure than for diastolic pressure in both the de Kort and Moreau studies. The effect was statistically significant in the range of 12-30 ug/dl in the Moreau paper, although the effect was not large at that level.

A more recent longitudinal study by Weiss et al. (1986) examined the blood-lead/blood-pressure relationship in 89 Boston policemen. This study also found a stronger correlation with systolic than diastolic blood pressure. Weiss' high-lead group had blood lead levels $\geq$ 30 ug/dl.

There are several recent general population studies, as well. Kromhout and Couland (1984) and Kromhout et al. (1985) studied 152 men, aged 57-67, drawn from the general population. They found a significant relationship between blood lead and blood pressure. However, the statistical significance of the findings decreased or disappeared after eliminating the highest blood-lead subject and after multiple regression analyses were conducted

that included other determinants of blood pressure, such as age and body mass. The authors concluded that blood lead is probably a less important determinant of blood pressure than age or body mass.

> "The above recent studies provide generally consistent evidence of increased blood pressure being associated with elevated lead body burdens in adults, especially as indexed by blood lead levels in various cohorts of working men. None of the individual studies provide definitive evidence establishing causal relationships between lead exposure and increased blood pressure. Nevertheless, they collectively provide considerable qualitative evidence indicative of significant associations between blood lead and blood pressure levels. Particularly striking are the distinct dose-response relationship seen for systolic pressure (correcting for age, body mass, etc.) by Moreau et al. and the findings of significant associations between blood lead and systolic pressure after extensive and conservative statistical analyses by Weiss et al. However, estimates of quantitative relationships between blood lead levels and blood pressure increases derived from such study results are subject to much uncertainty, given the relatively small sample sizes and limited population groups studied. Two larger-scale recent studies of general population groups, reviewed next, provide better bases for estimation of quantitative blood-lead blood-pressure relationships." (Addendum, p. A-10)

IV.A.1.c. Population Studies

Pocock et al. (1984) evaluated the relationships between blood lead level, hypertension and renal function in a clinical study of 7,735 middle-aged (aged 40-49) British men. (This study, conducted by the British government is also called the British Regional Heart Study.) In that article, the authors interpreted their findings as being suggestive of increased hypertension at elevated blood-lead levels (> 37 ug/dl), but not at the lower levels found typically in British men. However, more recent multiple regression analyses, adjusted for variation due to site, reported in Pocock et al. (1985) for the same data indicate highly statistically significant associations between both systolic ($p = 0.003$) and diastolic ($p < 0.001$) blood pressure and blood lead levels. Noting the small magnitude of the observed association and the difficulty in adjusting for all potentially relevant confounders, Pocock et al. (1985) cautioned against prematurely concluding that there is a causal relationship between body lead burden and blood pressure.

Several other studies of this relationship, discussed below, have been published using data from the Second National Health Assessment and Nutritional Evaluation Survey (NHANES II),* which provide careful blood lead and blood pressure measurements on a

* The NHANES II was a 10,000 person representative sample of the U.S. non-institutionalized population, aged 6 months to 74 years. The survey was conducted by the (U.S.) National Center for Health Statistics (NCHS) over a four-year period (1976-1980). The data base is available from NCHS and analyses of the lead-related data from it have been published before (e.g., Annest et al., 1982 and 1983; Mahaffey et al., 1982a and 1982b; Pirkle and Annest, 1984).

large-scale sample representative of the U.S. population and considerable information on a wide variety of potentially confounding variables, as well. The studies using the NHANES II data avoided the problems of selection bias, healthy worker effect, work place exposures to other toxic agents, and appropriate choice of controls, that had complicated or confounded many of the other studies (cf, Addendum to the Criteria Document, 1986; p. A-12ff).

Simple correlation analyses reported by Harlan et al. (1985) demonstrated statistically significant linear associations ($p < 0.001$) between blood lead concentrations and blood pressure (both diastolic and systolic) among males and females, aged 12-74 years. Controlling for many potentially confounding factors, multiple regression analyses showed the blood-lead/blood-pressure relationships remained significant for males but not for females.

Pirkle et al. (1985) conducted additional analyses on the NHANES II data. That study focused on white males, aged 40-59, to avoid the effects of collinearity with age, and because of less extensive NHANES II data being available for non-whites. In the subgroup studied, Pirkle et al. found significant associations between blood lead levels and blood pressure both in basic models and after including all the known factors previously established as being correlated with blood pressure. The relationship also held, with little change in the coefficient, when tested against every dietary and serological variable measured in the NHANES II.

> "No evident threshold was found below which blood lead level was not significantly related to blood pressure across a range of 7 to 34 ug/dl. The dose-response

relationships characterized by Pirkle indicate that large initial increments in blood pressure occur at relatively low blood-lead levels, followed by a leveling off of blood pressure increments at higher blood-lead levels. Pirkle et al. also found lead to be a significant predictor of diastolic blood pressure greater than or equal to 90 millimeters of mercury (mm Hg),* the criterion blood pressure level now commonly employed in the United States to define hypertension. Additional analyses were performed by Pirkle et al. to estimate the likely public health implications of the Pirkle findings concerning the blood-lead/blood-pressure relationship. Changes in blood pressure that might result from a specified change in blood lead levels were first estimated. Then coefficients from the Pooling Project and Framingham studies (Pooling Project Research Group, 1978; and McGee and Gordon, 1976, respectively)** of cardiovascular disease were used as bases: (1) to estimate the risk for incidence of serious cardiovascular events (myocardial infarction, stroke, or death) as a consequence of lead-induced blood pressure increases and (2) to predict the change in the number of serious outcomes as the result of a 37 percent decrease in blood lead levels for adult white males (aged 40-59 years) observed during the course of the NHANES II survey (1976-1980)." (Addendum, p. A-13).

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* Millimeters of mercury is the standard measure of blood pressure.

** These are discussed more fully in Section IV.A.4., below.

Schwartz (1985) expanded the Pirkle et al. analysis to include all men over 20, and examined the 20-44 and 45-74 age groups separately. In all three groups, lead was significant both in basic models and when tested against the much-larger variable list. Schwartz also showed that lead was significant even with the inclusion of a linear time-trend term.

> "Questions have been raised by Gartside (1985) and Du Pont (1986) regarding the robustness of the findings derived from the analyses of the NHANES II data as to whether certain time trends in the NHANES II data set may have contributed to (or account for) the reported blood-lead/blood-pressure relationships. . . However, neither the Gartside nor the Du Pont analyses adjusted for all of the variables that were selected for stepwise inclusion in the Harlan et al. (1985) and Pirkle et al. (1985) published studies." (Addendum, p. A-14)

Reanalyses of the NHANES II data by Schwartz (1986) showed that lead remained a significant factor even if a linear time trend was included in the regression and also remained significant in regressions that adjusted for all of the sites visited in NHANES II, which controls for both time (the sites were visited sequentially) and geographical variation. Schwartz also showed that the relationship held in all adult men, in men under 45, and in men over 45, as well as in the age group of Pirkle et al. These reanalyses were reviewed and accepted by EPA's external Science Advisory Board.

> "In order to more definitively assess the robustness of the Harlan et al. (1985) findings and, also, to evaluate possible time-trend effects confounded by variations in sampling sites, Landis and Flegal (1986) carried out further analyses for NHANES II males, aged 12-74, using a randomization model-based approach to test the statistical significance of the partial correlation between blood lead and diastolic blood pressure, adjusting for age, body mass index, and the 64 NHANES II sampling sites. The resulting analyses confirm that the significant association between blood lead and blood pressure cannot be dismissed as spurious due to concurrent secular trends in the two variables over the NHANES study period." (Addendum, p. A-14)

In addition, EPA has conducted a series of additional reanalyses of the NHANES II data to address the issue of "site" more definitively.

> "These unpublished analyses* confirm that the regression coefficients remain significant for both systolic and diastolic blood pressure when site is included as a variable in multiple regression analyses." (Addendum, p. A-15)

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* Available in the Central Docket Section of EPA. Docket number, ECAO-CD-81-2; documents numbered IIA.F.60, IIA.C.5, II.A.C.9, and IIA.C.11.

"Overall, the analyses of data from the two large-scale general population studies (British Regional Heart Study and U.S. NHANES II study), conducted both in this country and in Great Britain, collectively provide highly convincing evidence demonstrating small but statistically significant associations between blood lead levels and increased blood pressure in adult men. The strongest associations appear to exist for males aged 40-59 and for systolic pressure somewhat more than for diastolic. Virtually all of the analyses revealed positive associations for the 40-59 age group, which remain or become significant (at $p < 0.05$) when adjustments are made for geographic site. Furthermore, the results of these large-scale studies are consistent with similar findings of statistically significant associations between blood lead levels and blood pressure increases as derived from other recent smaller-scale studies discussed earlier, which also mainly found stronger associations for systolic pressure than for diastolic. None of the observational studies in and of themselves can be stated as definitively establishing causal linkages between lead exposure and increased blood pressure or hypertension. However, the plausibility of the observed associations reflecting causal relationships between lead exposure and blood pressure increases is supported by: (1) the consistency of the significant associations that have now been found by numerous independent

investigators for a variety of study populations; and (2) by extensive toxicological data (see below) which clearly demonstrate increases in blood pressure for animal models under well-controlled experimental conditions. The precise mechanisms underlying the relationships between lead exposure and increased blood pressure, however, appear to be complex, and mathematical models describing the relationships still remain to be more definitively characterized. At present, log blood-lead/blood-pressure (log PbB-BP) models appear to fit best the available data, but linear relationships between blood lead and blood pressure cannot be ruled out at this time. The most appropriate coefficients characterizing blood-lead/blood-pressure relationships also remain to be more precisely determined, although those reported by Landis and Flegal (1986) and those obtained by analyses adjusting for site appear to be the currently best available and most reasonable estimates of the likely strength of the association (i.e., generally in the range of 2.0-5.0 for log PbB versus systolic and 1.4 to 2.7 for log PbB versus diastolic blood pressure)."

"Blood lead levels that may be associated with increased- blood pressure also remain to be more clearly defined. However, the collective evidence from the above studies points toward moderately elevated blood-lead levels ($\geq$ 30 ug/dl) as being associated most clearly with blood pressure increases, but certain evidence

(e.g., the NHANES II data analyses and the Moreau et al. study results) also indicates significant (and apparently stronger) relationships between blood pressure elevations and still lower blood lead levels that ranger possibly, to as low as 7 ug/dl. This may be supported by several animal studies, discussed below, that also find hypertension most consistently related to relatively low exposure levels but over relatively long exposure periods."

"The quantification of likely consequent risks for serious cardiovascular outcomes, as attempted by Pirkle et al. (1985), also remains to be more precisely characterized. The specific magnitudes of risk obtained for serious cardiovascular outcomes in relation to lead exposure, estimated on the basis of lead-induced blood pressure increases, depend crucially upon: the form of the underlying relationship and size of the coefficients estimated for blood-lead/blood-pressure associations; lead exposure levels at which significant elevations in blood pressure occur; and coefficients estimating relationships between blood pressure increases and specific, more-serious cardiovascular outcomes. As noted above, uncertainty still exists regarding the most appropriate model and blood-lead/blood-pressure coefficients, which makes it difficult to resolve which specific coefficients should be used in attempting to project more serious cardiovascular outcomes. Similarly, it is difficult to

> determine appropriate blood-lead levels at which any selected coefficients might be appropriately applied in models predicting more serious cardiovascular outcomes. Lastly, the selection of appropriate models and coefficients relating blood pressure increases to more serious outcomes is also fraught with uncertainty...
> Further analyses of additional large-scale epidemiologic data sets may be necessary in order to determine more precisely quantitative relationships between blood lead levels and blood pressure, and more serious cardiovascular outcomes as well."
>
> "The findings discussed here, while pointing toward a likely causal effect of lead in contributing to increased blood pressure, need to be placed in broader perspective in relation to other factors involved in the etiology of hypertension. The underlying causes of increased blood pressure or "hypertension" (diastolic blood pressure above 90 mm Hg), which occurs in as many as 25 percent of Americans, are not yet fully delineated. However, it is very clear that many factors contribute to development of this disease, including hereditary traits, nutritional factors and environmental agents." (Addendum, p. A-15 to A-18)

The contribution of lead, compared to many other factors such as age, body mass, and smoking, appears to be relatively small, but the findings have stayed robust in the face of repeated reanalyses and specifications of the models.

IV.A.2. Mechanisms Potentially Underlying Lead-Induced Hypertension Effects

"This section [briefly summarizes] plausible biochemical-physiological mechanisms by which lead potentially influences the cardiovascular system to induce increased blood pressure, followed by [a short discussion] of experimental evidence concerning the contribution of lead exposure to the development of hypertension.

"Blood pressure is determined by the interaction of two factors: cardiac output and total peripheral resistance. An elevation of either or both results in an increase in blood pressure. A subsequent defect in a critical regulatory function (e.g., renal excretory function) may influence central nervous system regulation of blood pressure, leading to a permanent alteration in vascular smooth muscle tone which sustains blood pressure elevation. The primary defect in the pathophysiology of hypertension is thought to be due to alteration in calcium binding to plasma membranes of cells; this change in calcium handling may in turn be dependent upon an alteration in sodium permeability of the membrane (e.g., Hilton, 1986). This change affects several pathways capable of elevating pressure: one is a direct alteration of the sensitivity of vascular smooth muscle to vasoactive stimuli; another is indirect, via alteration of neuroendocrine input to vascular smooth muscle (including changes in renin secretion rate)." (Addendum, p. A-18)

IV.A.2.a. Role of Disturbances in Ion Transport by Plasma Membranes

"Many stimuli activate target cells in the mammalian body via changes in ion permeabilities of the plasma membrane, primarily for sodium, potassium, and calcium ions; the change in calcium ion concentration is the primary intracellular signal controlling muscle contractions, hormone secretion, and other diverse activities... For calcium, there is a membrane potential-dependent sodium/calcium exchange pump which extrudes one calcium ion in exchange for three sodium ions. In addition, there are calcium ATPase pumps located at cell membranes and at intracellular membrane storage sites (endoplasmic reticulum and mitochondria)... The ion interacts with several calcium-binding proteins which, in turn, activate cell contractile or secretory processes.

"It has been postulated that sodium pump inhibition by some endogenous factor (thought to be a hormone) would be ultimately causatory for development of both essential and volume-expanded hypertension by affecting vascular tone or resistance.

"If lead exposure could be shown to affect sodium transport (which, then, indirectly alters vascular resistance") or to directly affect vascular resistance (by changing calcium ion permeability or transport), it could contribute to the development of hypertension...

> Abundant experimental evidence... indicates that lead affects both; that is, lead inhibits cell membrane-bound sodium-potassium-ATPase as well as interferes with normal processes of calcium transport across membranes of various tissue types... Lead acts to alter sodium balance and calcium-activated cell activities of vascular smooth muscle. Changes in either or both of these could be expected to produce changes in blood pressure regulation." (Addendum, p. A-18 to A-19)

IV.A.2.b. Role of Renin-Angiotensin in Control of Blood Pressure and Fluid Balance

One major endogenous factor regulating total peripheral resistance of the vascular smooth muscle is angiotensin II (AII), a small peptide generated in plasma via the action of a renal hormone, renin.

> "The renin-angiotensin system has a major influence on regulation of blood pressure, [both directly and indirectly. It directly affects vascular smooth muscles to increase vasoconstriction and it indirectly increases total peripheral resistance by affecting the discharge rate of sympathetic neurons.] For this reason, investigators interested in hypertension have studied the system in detail. Because renal disease may be an important initiating event in subsequent development of hypertension and because lead is an important renal toxicant, some investigative reports of patients with lead intoxication have evaluated blood pressure changes

and changes in the renin-angiotensin system." (Addendum, p. A-20 to A-21)

However, the results of these studies have been contradictory. The few human studies have shown depressed, increased and unaltered renin activity in lead intoxicated men. The studies may not be comparable because some used men with chronic sub-clinical lead exposure as compared to chronic heavy lead exposure, and some subjects had exposure which would be considered "normal".

In addition, there have been animal and experimental studies, investigating the cardiovascular effects of both acute and chronic exposure to lead.

> "Lead injected intravenously in dogs and rats, at doses as low as 0.1 mg/kg (whole blood lead < 5 ug/dl and renal lead of 1.2 ug/g) produced over the next several hours significant increases in plasma renin activity (PRA) and in excretion of sodium, other cations, and water (Mouw et al., 1978)... The increased sodium excretion could be attributed to decreased sodium reabsorption. The mechanism of lead's action on tubular reabsorption was not determined,... nor was the mechanism by which lead increased renin secretion.
>
> "In a subsequent study, Goldman et al. (1981) found that the rise in PRA after acute lead injection was not due to increased renin secretion in six of nine dogs; rather there was elimination of hepatic renin clearance, without evidence for other interference in liver function. In the remaining three dogs, renin secretion increased; this was thought to be due to lead activation of normal mechanisms

for renin secretion, although none of the classic pathways for influencing renin secretion were altered. The authors postulated that lead might produce alterations in cytosolic calcium concentration in renin-secreting cells... The authors also postulate that there may be multiple actions of lead on the renin-angiotensin system which may help explain confusion about the ability of lead to cause hypertension. At certain exposure conditions, there could be elevated PRA without simultaneous inhibition of angiotensin-converting enzyme, thereby contributing to hypertension, while higher doses or longer exposure might inhibit the converting enzyme and thereby cause loss of hypertension...

"The literature of experimental findings of lead-induced changes in the renin-angiotensin system and blood pressure in animals is complicated by apparently inconsistent results when comparing one study to another. All studies report changes in the renin-angiotensin system, yet some studies fail to find an effect on blood pressure and others do report hypertension. Doses and exposure periods employed vary widely, but in general, hypertension is observed most consistently with relatively low doses over relatively long exposure periods...

"Perry and Erlanger (1978) found that chronically feeding rats either cadmium or lead at doses of 0.1, 1.0, or 5.0 parts per million (ppm) produces statistically significant increases in systolic blood pressure...

The implications for human populations exposed to very low doses of these metals were pointed out. Victery et al. (1982a) reinvestigated the question, using lead doses of 100 and 500 ppm administered in the drinking water to rats beginning while the animals were in utero and continuing through six months of age. At 3-1/2 months of age, the male rats drinking 100 ppm of lead first demonstrated a statistically significant increase in systolic blood pressure; this difference persisted for the remainder of the experiment. Animals drinking 500 ppm had lower pressures, which were not significantly different from controls. Female rats drinking 100 ppm did not demonstrate pressure changes. At termination of the experiment PRA was significantly decreased by 100 ppm lead exposure, but not 500 ppm... There was a dose-dependent decrease in the ratio of [angiotensin II to plasma renin activity] for lead-exposed rats. Renal renin was depressed in lead-exposed animals. The hypertension observed in these animals was not secondary to overt renal disease (as opposed to an effect on renal cell metabolism), as evidenced by a lack of changes in renal histology and plasma creatinine.

"Victery et al. (1983) examined changes in the renin-angiotensin system of rats exposed to lead doses of 5, 25, 100, or 500 ppm during gestation until one month of age. All had elevated plasma renin activity, while those at 100 and 500 ppm also had increased renal renin

concentration. Lead-exposed animals... secreted less renin than control animals. It appears that lead has two chronic effects on renin secretion, one inhibitory and one stimulator; the magnitude of effect on PRA reflects the dose and timing of the lead exposure as well as the physiological state of the animal.

"In another study, Victery at al. (1982b) reported that rats fed 5 or 25 ppm lead for five months (blood lead of 5.6 and 18.2 ug/dl, respectively) did not develop hypertension but at 25 ppm had significantly decreased PRA. Both groups of animals had a decrease in the AII to PRA ratio. Thus, lead exposure at levels generally present in the human population caused observable effects in renin synthesis." (Addendum, p. A-23 to A-25)

IV.A.2.c. Effects of Lead on Vascular Reactivity

"Piccinini et al. (1977) and Favalli et al. (1977) studied the effects of lead on calcium exchanges in the isolated rat tail artery; lead in concentrations of up to 15 umol in vitro produced contractions which required the presence of calcium in the perfusion solution. Therefore, calcium influx was not affected by lead... Tissue calcium content was increased...

"Tail arteries obtained from the hypertensive rats in the study performed by Victery et al. showed an increased maximal contractile force when tested in vitro with the alpha-adrenergic agents norepinephrine and

methoxamine (Webb et al., 1981). This finding is apparently related to an increase in the intracellular pool of activator calcium in the smooth muscle cells in the artery. This change may also be responsible for decreased relaxation of the muscle after induced contractions.

"In vivo tests of cardiovascular reactivity in rats exposed to 50 ppm lead (blood lead 38.4 ± ug/dl) for 160 days were performed by Iannaccone et al. (1981). [This study showed] significant increases in systolic and diastolic pressure [related to lead exposure, as well as significant increases in the blood pressure response to noradrenalin]. The data suggest that the lead-related increase in arterial pressure is due at least in part to greater sympathetic tone, with the metal affecting neural control of blood pressure." (Addendum, p. A-26 to A-27)

IV.A.2.d. Effects of Lead on Cardiac Muscle

Lead has been hypothesized to contribute to cardiomyopathy and to have cardiotoxic properties.

"Kopp et al. (1978) developed an in vitro system for monitoring the cardiac electrical conduction system (electrocardiogram or ECG) and systolic tension, and demonstrated that in vitro lead (3 x 10-2 mM) or cadmium (3 x 10-2 mM) depressed systolic tension and prolonged the P-R interval of the ECG... [In a later study,] hearts obtained from rats exposed to low levels of cadmium and/or lead (5 ppm) for 20 months were found to have... changes in the heart's electrical conduction system (Kopp et al., 1980) with significant prolongation of the P-R interval. . .

"Williams et al. (1983) suggested that much of the negative effect of lead on cardiac tissue and ECG abnormalities can be related to lead's interference with calcium ion availability and/or membrane translocation. In addition, even those lead exposure-related effects that appear to occur through autonomic nerves may be understood in terms of effects on calcium ion, which is required for neurotransmitter release...

"Prentice and Kopp (1985) examined functional and metabolic responses of the perfused rat heart produced by lead with varying calcium concentrations in the perfusate. Lead altered the spontaneous contractile activity, spontaneous electrical properties and metabolism of the heart tissue. The exact mechanisms were not completely resolved but did involve disturbances in cellular calcium metabolism, although not by any single mechanistic model...

"In addition, hearts perfused with 30 uM lead had reduced coronary blood flow, presumably by lead acting to directly constrict the vascular smooth muscle or by interference with the local metabolic stimuli for vasodilatation. Increases in perfusate calcium concentration partially reversed this effect, although at the highest calcium levels (5.0 mM) coronary blood flow was again reduced. These authors concluded that their present findings were consistent with those of others which

showed increased vascular reactivity and that the chronic lead exposure-related changes in blood pressure may be related to localized actions of lead on vascular beds and arterial smooth muscle." (Addendum, p. A-27 to A-29)

IV.A.2.e. Summary of Lead-Related Effects on the Cardiovascular System

"Blood pressure is regulated and affected by many interactive forces and control systems; some of these have been shown to be affected by lead exposure. Understanding of the effects of lead on each system is still preliminary, but sufficient evidence indicates that changes which occur in the presence of lead can promote the development of hypertension... Although the exact mechanisms involved in lead-induced changes in renin secretion rate have not been examined, it is likely that lead could be affecting the cystosolic free calcium ion of [some] cells... After lead enters [these] cells, lead could enhance or block calcium exit via sodium/calcium exchange pumps, or increase or decrease the intracellular sequestration of calcium in storage compartments...

"The changes in vascular reactivity which have been reported in animals chronically exposed to lead are probably the key finding which can lead to an understanding of how lead can contribute to the development

of hypertension. The vascular smooth muscle changes are necessary and sufficient in themselves to account for the increase in blood pressure and the fact that these changes are observed in animals exposed to relatively low lead levels makes it increasingly important to evaluate these findings in additional experimental studies. There may be additional changes in the entire sympathetic neural control of vascular tone which acts to amplify the contractile response to any endogenous vasoconstrictor substance." (Addendum, p. A-29 to A-30)

IV.A.3. Cardiovascular Disease Rates and Water Hardness

Since Kobayashi's study (1957) was highlighted by Schroeder (1958), studies in several countries have evaluated the relationship between drinking water characteristics and death from hypertensive, ischemic or arteriosclerotic heart diseases, and in particular the relationship between cardiovascular disease (CVD) rates and water hardness.* Generally, the studies covering large geographic regions have shown a significant inverse relationship between the hardness of local drinking water and local CVD rates. However, not all the studies, most commonly not the studies involving smaller geographic regions, evidence the same consistency of effect.

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* Water hardness is determined by the relative amount of dissolved solids, primarily calcium and magnesium, in it. It is expressed as the equivalent amount of calcium carbonate ($CaCO_3$) that could be formed from the calcium and magnesium in solution. For a fuller discussion of this issue, see Chapters II and V of this document. Generally, water < 60 mg/l as $CaCO_3$ is considered 'soft'. Most studies investigating CVD and water hardness have focused upon systems with very soft water, i.e., $\leq$ 40 mg/l as $CaCO_3$.

The U.S. National Research Council, of the National Academy of Sciences, convened a panel of experts in the late 1970s to review this issue. The panel concluded that the findings were "strikingly" equivocal and called for additional research (Angino, 1979). Since then, many additional reviews have attempted to resolve the inconsistencies in the data; none has succeeded. Two noteworthy reviews (Comstock, 1979; Comstock, 1985) apply rigorous statistical and epidemiologic tests to the literature on the relationship between water hardness and CVD. This section contains a very brief survey of the nature of these studies, plus a suggested explanation for the inconsistent findings.

IV.A.3.a. Studies of Cardiovascular Disease and Water Hardness

The largest category of studies is the intra-national studies, where various population units within several different countries were examined. Generally, these studies have found a statistically significant inverse relationship between CVD and water hardness, and account for the phrase: 'soft water, hard arteries'.

A second category of studies -- geographic units comprising states or provinces -- in general show a similar but weaker negative association between CVD mortality and water hardness.

Studies of selected communities that are not in the same geographic unit and that have different water hardnesses have been least likely to show consistent findings linking CVD and water parameters; the inconsistencies of findings have included associations with water hardness that were in opposite directions for the two sexes or for different categories of cardiovascular illnesses, as well as statistically insignificant associations.

Fewer of the studies conducted in the past decade have found the significant negative associations that were found in some of the earlier papers.

Interest in identifying a 'water factor' that influences heart disease has continued because CVD rates vary significantly by geographic regions (Sauer, 1980). Water parameters also tend to portray regional patterns and, therefore, could offer a tenable explanation for the geographic variation in mortality. Further, water treatment costs are lower than medical treatment costs.* At the water system level, reducing the presence of a harmful substance or correcting the deficiency of a protective one is both relatively easily done and effective in reaching large numbers of people.

There are many plausible mechanisms for recognizing an effect from the constituents and parameters of water upon heart disease. The presence or selective absence of trace elements are the most likely sources of an effect on health related to corrosive water. Or soft water may initiate heart disease, or soft water parameters added onto other factors that predispose a person to heart disease may be sufficient to push him/her over the threshold to symptomatic illness, or it may exacerbate a pre-existing condition, either recognizable or asymptomatic. Soft water may contain harmful contaminants or hard water may contain protective elements lacking in soft water. Most probably, some combination of these scenarios accounts for the association.

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* For instance, treatment to increase water hardness costs $1-2 per person per year as opposed to one heart attack, which costs $65,000 per case.

Generally, there are three broad classes of mechanisms by which water can be postulated to affect CVD in either a causative or detrimental manner.

1) Soft water is deficient in some major cations that are protective to the cardiovascular system and that are abundant in hard water. The most likely of these are calcium and magnesium, which are the major components of hardness in water. Some epidemiological studies (e.g., Abu-Zeid, 1979; though not all, e.g., Miller et al., 1985) have shown magnesium to be a strong factor in cardiovascular health.

2) Protection of the cardiovascular system by specific minor cations or trace metals that are more abundant in hard water than soft water. Of these, lithium, chromium, manganese, selenium, vanadium, and strontium are possible contributors. All, with the possible exception of vanadium, are essential micronutrients and could have plausible involvement with CVD rates.

3) Soft water leaches toxic metals from the distribution system. Studies relating water hardness to CVD have considered especially three potential toxins: cadmium, copper, and lead. Copper is perceived as the least likely of the three, and strong evidence exists linking both lead and cadmium to hypertension. All three metals are corrosion by-products. (It is interesting, in this light, to note that water is a more significant source of lead in Britain than the U.S. and the hard-water/CVD association appears more robust there.)

Overall, the two constituents most seriously considered in current analyses are the protective role of magnesium (less common in soft water than in hard water) and a detrimental effect from increased sodium (which is often added in water softening units).*

IV.A.3.b. Lead, Soft Water and Cardiovascular Disease

While the geographic variation in CVD probably relates to several factors and the interrelationships between them, the new studies relating blood lead levels to blood pressure could help resolve some of the inconsistent correlations between cardiovascular disease and soft drinking water. Lead occurs in drinking water as a corrosion by-product, that is, as a result of the action of corrosive water upon the materials (pipes and particularly solder) of the distribution and residential plumbing systems.** Hardness/softness of water is one measure of a water's potential corrosivity, but because water chemistry is very complicated, no single measure is an adequate predictor of a specific water's actual corrosivity or of the presence of corrosion by-products, including lead (cf, for instance, Patterson, 1981).

Relatively few of the studies investigating the relationship between water hardness and CVD rates have considered the presence

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* It has been suggested that the relatively recent increase in sodium intake in populations in hard water areas may be negating the advantages once associated with those hard water areas (Comstock, 1985).

** For a fuller discussion of these studies, see Section II.A., above, or Section V.A., below.

of trace metals at all (e.g., Sharrett et al., 1984) or the water sampling procedures have not addressed the occurrence phenomena of corrosion by-products.* Often, the water data represent lead levels in the distribution system (e.g., Shaper et al., 1980; or Craun and McCabe, 1975, which uses data from the 1969 Community System Survey, presented in McCabe et al., 1970); or tap water typical of the distribution system (i.e., fully flushed); or tap water averaged with distribution system water (e.g., Greathouse and Craun, 1978; or Greathouse and Osborne, 1980; which use data from the First National Health and Nutrition Augmentation Survey, in which one random daytime tap sample was averaged with 12 monthly samples from the water supply).

In the few studies that did consider exposure to corrosion by-products, other problems have arisen. In Calabrese and Tuthill (1978), for instance, the lead levels in the two communities studied were not statistically significantly different. Other studies have not withstood rigorous statistical testing (e.g., Hewitt and Neri, 1980).

Considering the data linking blood pressure (and CVD) to exposure to lead, the studies correlating CVD with water hardness may be imperfectly measuring the relationship between soft drinking water and the presence of lead (and/or cadmium). In other words, the studies that have found a relationship between soft water and heart disease may actually have evaluated systems with higher-than-

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* That is, contamination of drinking water that occurs due to the corrosive action of the water upon the materials of the plumbing systems. Several of the trace constituents of concern, including lead, copper and cadmium, are corrosion by-products. This issue is discussed in Chapter II of this document.

average lead levels, resulting from softer water leaching more lead from the plumbing. However, while softer water is more likely to contain higher lead (or cadmium) levels, the factors related to lead contamination are very complicated. Soft water alone is not predictor of lead levels, and therefore, not all systems with soft water have higher metals. This may account for some of the inconsistent findings.

Another factor is that the major elements of hard water are magnesium and calcium which can be easily absorbed in the small intestine and therefore may compete with lead for some common transport system or metabolic interactions, as explained by Conrad and Barton (1978) and Mahaffey and Rader (1980). The relationship between calcium intake and lead absorption is not yet clear. Some early studies (e.g., Aub, 1935) indicated that increased calcium intake could reduce blood lead levels; other studies have shown more complicated relationships between calcium intake and lead absorption and retention, including lead's effect upon calcium metabolism (Six and Goyer, 1970 and 1972; Quarterman et al., 1978; also cf the discussions on lead and calcium in both the Criteria Document, 1986, p. 10-44 to 10-48, and its Addendum, p. A-18ff).

IV.A.4 Benefits of Reduced Cardiovascular Disease: Reductions in Hypertension and Related Morbidity and Mortality

On the basis of the data discussed above on the association between lead body burden and increased blood pressure in adult males, this analysis assumes that reducing lead in drinking water could reduce blood lead levels, which in turn could reduce blood

pressure and the number of individuals with hypertension. A reduction in hypertension would have direct benefits from reduced medical treatment expenditures. More important, however, are the related benefits in the form of reduced cardiovascular disease associated with elevated blood pressure. This section describes the methods used to estimate the benefits associated with lowering blood pressure, including estimating the reductions in morbidity and mortality.

Estimating the reduction in hypertension and cardiovascular disease requires several steps. The first is to estimate the impact of the reduction of lead levels in drinking water on levels of lead in adults' blood. For that, the occurrence data and water-lead to blood-lead equations described in Chapter II were combined with the regression analyses of the NHANES II data discussed previously. In each case, the blood lead levels in the NHANES II data were first adjusted to reflect reductions in environmental lead contamination that have occurred since the time of the survey.

To calculate the cardiovascular benefits, logistic regression equations were used to predict how reducing exposure to lead in drinking water could affect the number of hypertensives in the U.S. population. These estimates cover only males aged 40 to 59, because the effect of lead on blood pressure appears to be stronger for men and because the correlation between blood pressure and age is much smaller in this age range, reducing the potential for confounding due to the correlation between blood lead and age. The estimates of reduced cases of cardiovascular and

cerebrovascular disease rely upon (1) site-adjusted coefficients from analyses of the NHANES II data relating blood lead levels to increases in blood pressure* and (2) coefficients relating blood pressure increases to more serious cardiovascular disease outcomes, based on data from the Framingham Study and Pooling Project (1976), discussed below.

The estimates of myocardial infarctions, strokes and deaths are further restricted to white men, aged 40-59, because the NHANES data contain insufficient observations on non-whites to evaluate the form of the relationship among non-whites. This severely limits this analysis because both blood pressure and blood lead levels are higher among non-whites than whites.**

The fact that other sources of lead, especially gasoline, would slowly decline even without new EPA drinking water standards created a slight complication. Because gasoline lead levels fall over time as unleaded gasoline replaces leaded, the difference in blood lead levels resulting from this rule will change over time. The estimates in this report account for both the reduction

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* The specific coefficients and the basis for their derivation are described in the Addendum to the Criteria Document, 1986, which is included in Volume 1 of that publication. The issue of site adjustment is summarized briefly on p. IV-10ff of this document.

** In other EPA analyses, e.g., Methodology for Valuing Health Risks of Ambient Lead Exposure US-EPA 1986a), less conservative assumptions have been made -- i.e., assuming that the same cardiovascular risks apply to black men as to white men of the same age (cf., pages 5-5 ff). Extending this current analysis to black men, even using the same coefficients and assumptions as for white men, would raise the estimated benefits.

in blood pressure over the past decade and reductions in some other sources of lead. This model served EPA also in its analytical efforts supporting the most recent phasedown in the amount of lead permitted in leaded gasoline; it is discussed more fully in The Costs and Benefits of Reducing Lead in Gasoline (US-EPA, 1985b) . The coefficients were adjusted for site-variation as discussed in the Addendum to the Criteria Document (US-EPA, 1986; p. A-13ff).

IV.A.4.a. Hypertension

Based upon the studies relating lead exposure to blood pressure, discussed above, estimating the change in the number of cases of hypertension was straightforward: the logistic regression coefficients from Pirkle et al. (1985) adjusted for site were applied to the NHANES II data to predict the changes in the numbers hypertensives as a result of reducing the Maximum Contaminant Level (MCL) for lead in drinking water. In this analysis, EPA estimates that there would be 130,000 fewer cases of hypertension among males aged 40-59 in sample year 1988 as a result of this rule. The change due to this proposed regulation was calculated by subtracting the number at the new lead level from the number at the original lead level. This estimate covers only males aged 40 to 59, but includes non-whites as well as whites.

IV.A.4.b. Myocardial Infarctions, Strokes, and Deaths

Estimating the impact of reduced blood pressure on morbidity and mortality required several additional steps. Using the NHANES

II data and the site-adjusted regression coefficients described earlier, we simulated the changes in individual blood-pressure levels due to reductions in lead from drinking water. Coefficients from two large studies of cardiovascular disease were then used to estimate changes in the numbers of first-time myocardial infarctions, first-time strokes, and deaths from all causes.

The relationships between blood pressure and cardiovascular diseases have been established by several large, long-term epidemiological studies. The classic study, which was important in establishing cholesterol as a major factor in the risk of heart disease, was the Framingham study (McGee et al., 1976). Extensive analyses of these data have yielded estimates of cardiovascular risks associated with several variables, including blood pressure. Figure IV-1 shows the age-adjusted rates of death and heart attacks as functions of blood pressure from that study.

In the 1970s, the National Institutes of Health funded the Pooling Project (The Pooling Project Research Group, 1978), which combined the Framingham data with data from five other long-term studies to improve the accuracy of the risk coefficients for heart attacks. The Pooling Project tested the Framingham coefficients against the other study results and found that their predictive power was good. It then analyzed the first occurrence of myocardial infarctions in white men who entered the studies at ages 40 to 59 and who were followed for at least 10 years. The estimates of the numbers of first-time myocardial infarctions in this study employ the Pooling Project’s coefficients.